Abdominal Imaging

Compiled by;

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- Pancreatic Transplantation
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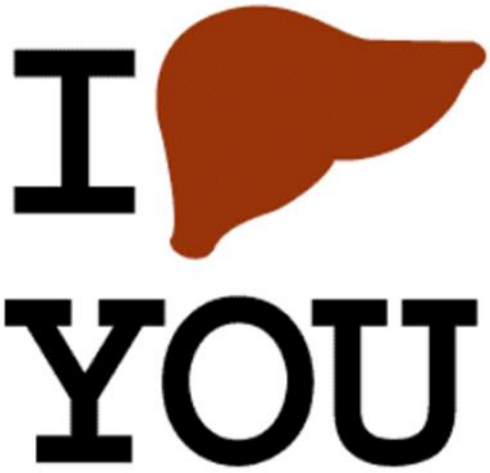
LIVER



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- **Lier Vascular Anatomy and Variants**
- **Imaging Techniques**
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- **Focal Cystic Lesions**
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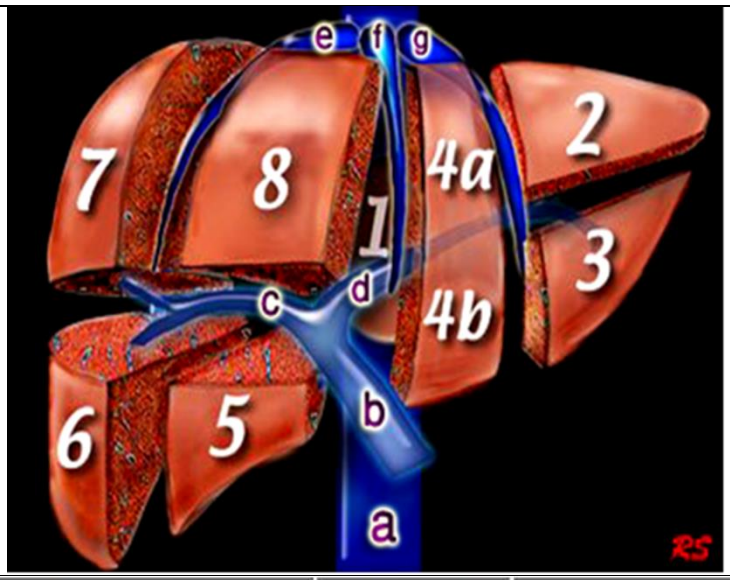
- **Perfusion Disorders**
- **Trauma**

Liver Segmental Anatomy



Gold Smith and wood burne Classification (1957)	Couinaud Classification (1957)	Bismuth Classification (1982)
Caudate lobe	Segment I	Segment I
left lateral segment		
• Superior subsegment	Segment II	Segment II
• Inferior subsegment	Segment III	Segment III
Left medial segment		
• Superior subsegment	Segment IV	Segment IV a
• Inferior subsegment		Segment IV b
Right anterior segment:		
• Superior subsegment	Segment V	Segment V
• Inferior subsegment	Segment VIII	Segment VIII
Right posterior segment:		
• Superior subsegment	Segment VI	Segment VI
• Inferior subsegment	Segment VII	Segment VII

❖ Couinaud and modified by Bismuth:

- is based on the three-dimensional concept that all hepatic segments except for the caudate lobe are defined by three vertical scissurae and a single transverse scissura

Role of hepatic veins => Vertical plane

- The middle hepatic vein divides the liver into right and left hemilivers.
- The right hepatic vein divides the right hemiliver into anterior (8&5) and posterior (7&6) segments.
- The left hepatic vein divides the left hemiliver into medial (4 A&B) and lateral (2&3) segments.

Role of portal veins => Transverse plane

- Each of these segments is divided into superior and inferior segments by a transverse line drawn through the right and left portal branches.

Gall bladder:

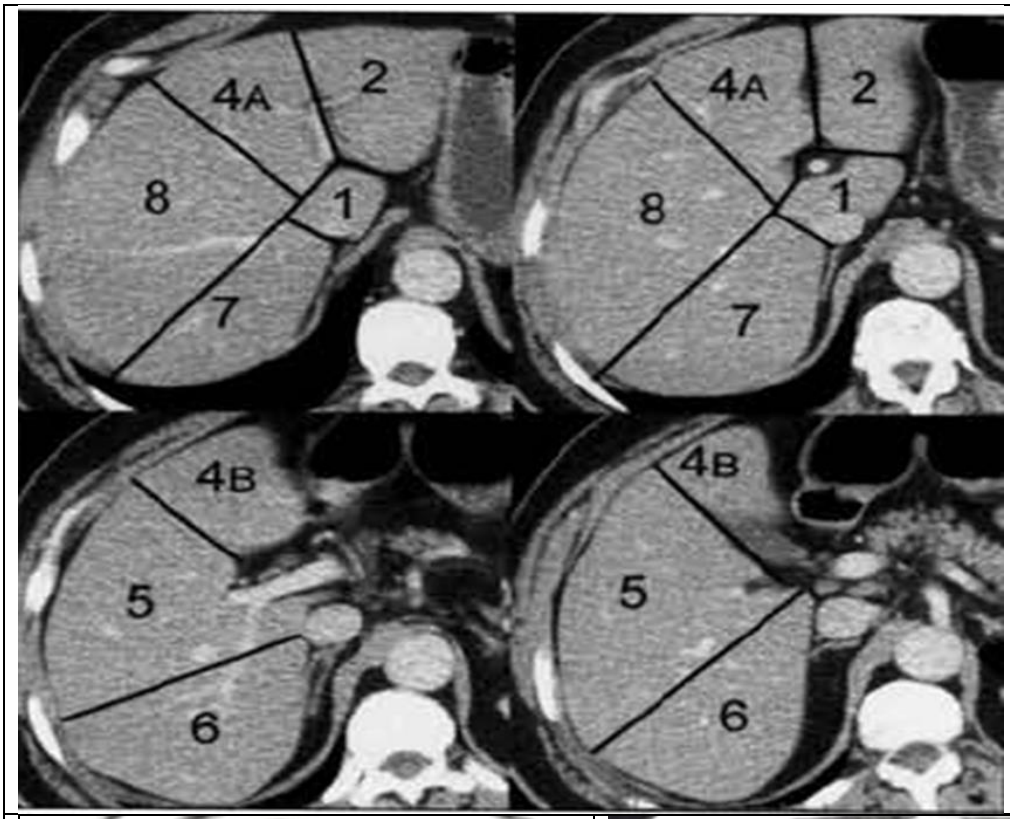
- Indicate position of main fissure between right and left liver lobes
- Separates segment IVB and segment V

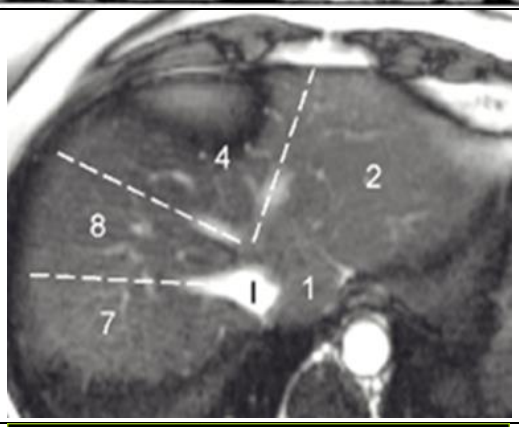
Falciform ligament:

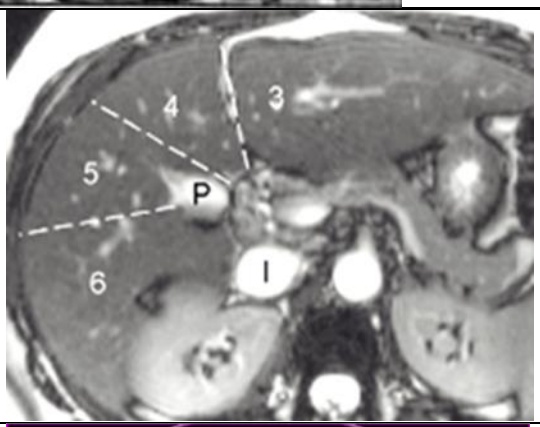
- Umbilical part of left portal vein, and cauded by ligamentum teres, separates left lateral segment II and III from left medial segment IV

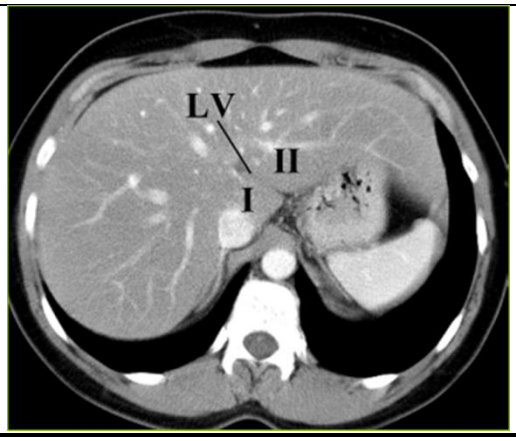
Ligamentum Venosum:

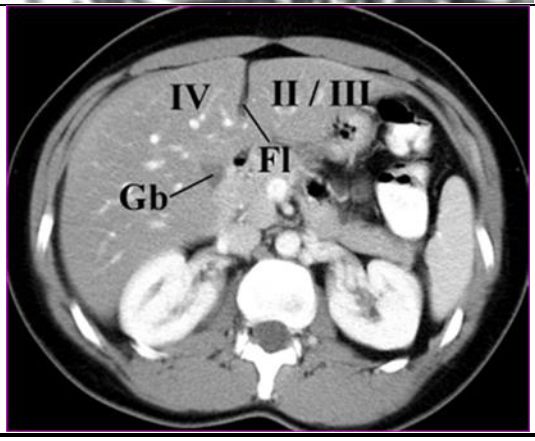
- Separates segment II anteriorly and caudate lobe (Segment I) posteriorly

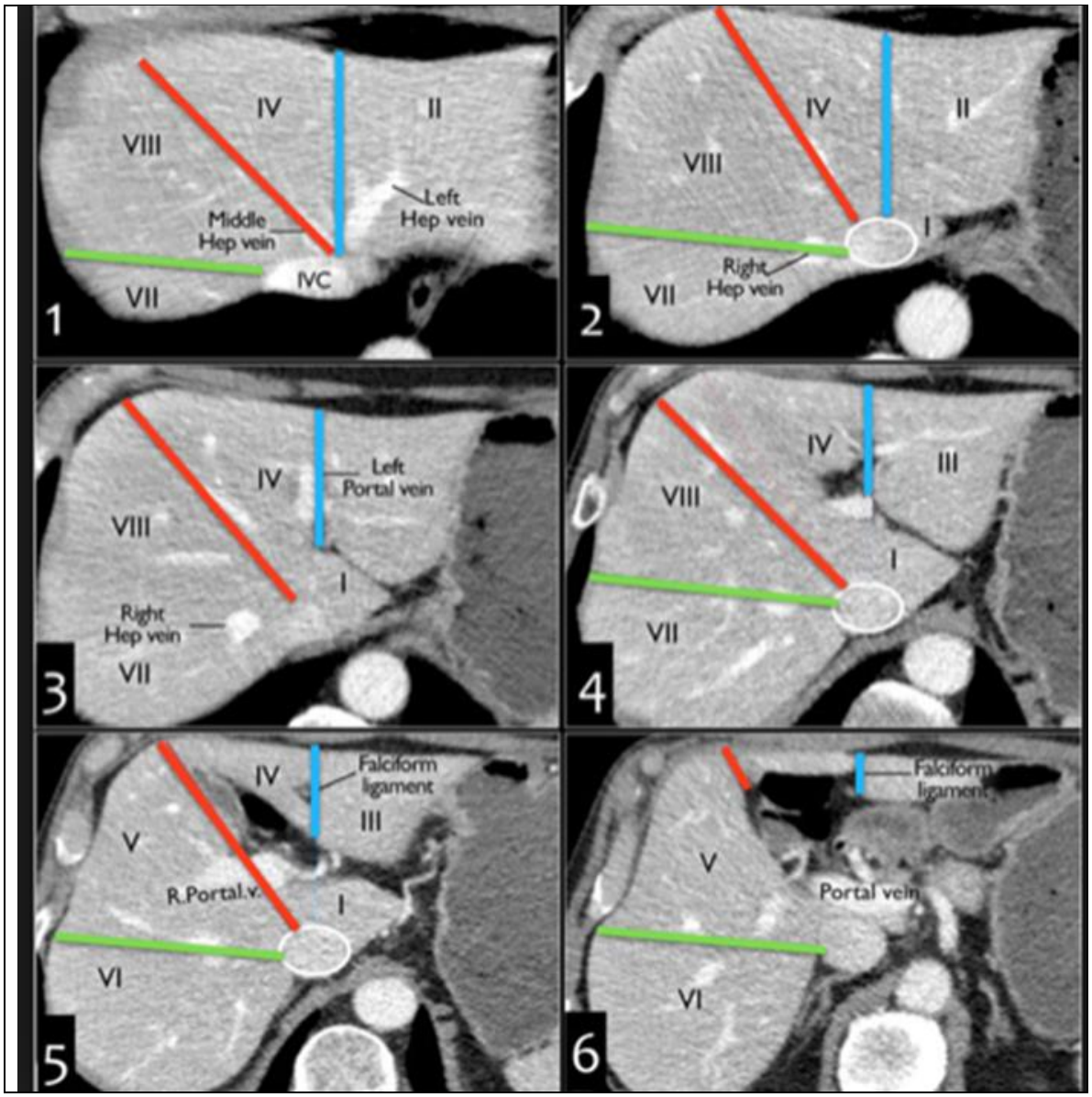

4A
2
8
1
7
4A
2
8
1
7
4B
5
6
4B
5
6

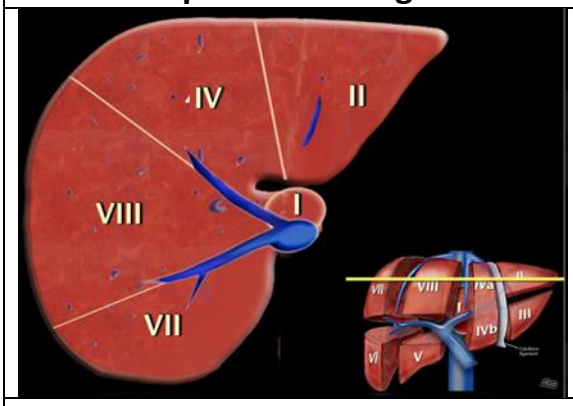
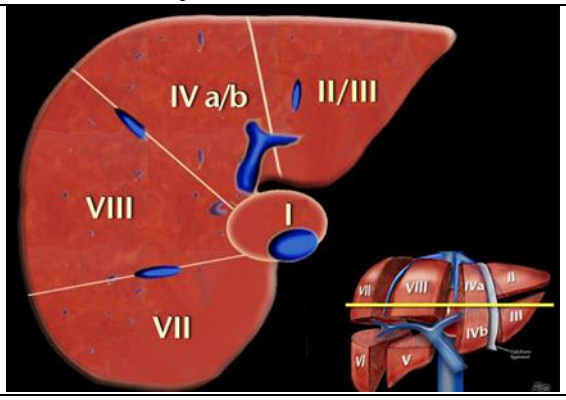
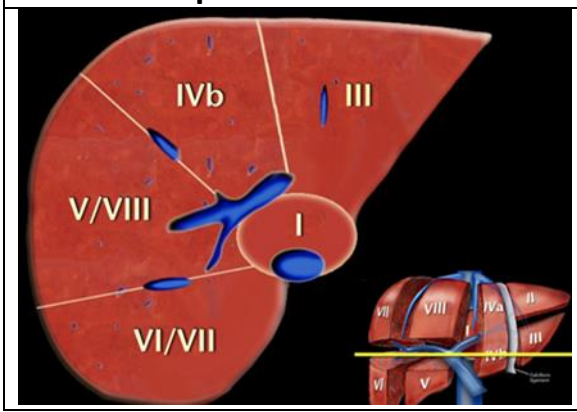
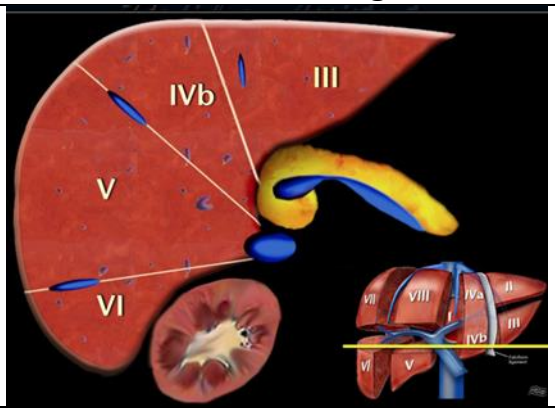

4
2
8
I
1
7


4
3
5
P
6
I


LV
II
I


IV
II / III
Fl
Gb


IV
II
VIII
Left
Hep vein
Middle
Hep vein
IVC
VII
1
IV
II
VIII
I
Right
Hep vein
VII
2
IV
Left
Portal vein
VIII
I
Right
Hep vein
VII
3
IV
III
VIII
I
VII
4
IV
Falciform
ligament
III
V
R.Portal.v.
I
VI
5
Falciform
ligament
V
Portal vein
VI
6

Level of superior liver segments	Level of LT portal vein
	
Level of RT portal vein	**Level of inferior liver segments**
	

Hepatic Vascular Anatomy and Variants

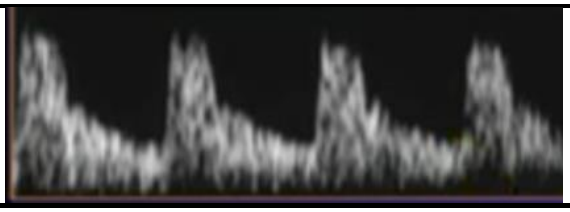
A distinctive feature of the liver is its unique dual blood supply:

- **Hepatic artery (25% of vascularization)**
- **Portal vein (75% of vascularization)**

A- Hepatic Artery

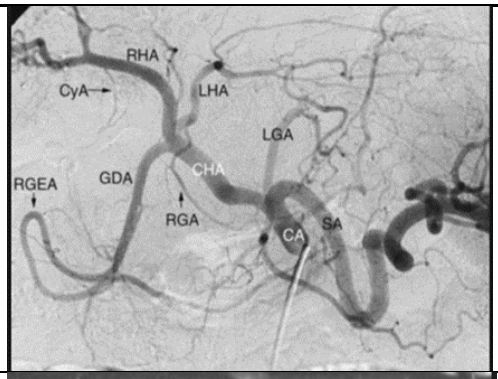
❖ **Normal Doppler Waveform:**

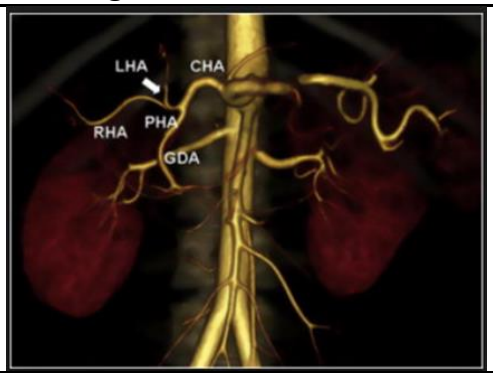
- Continuous hepatopetal flow
- Low resistance flow pattern
- RI = 0.5 – 0.7

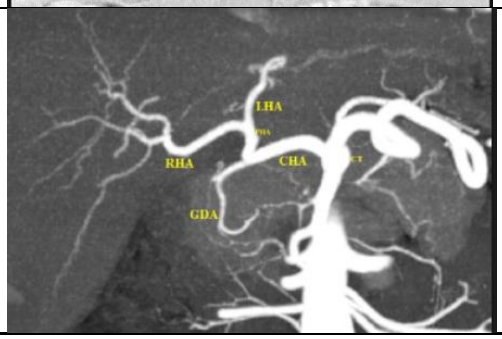


❖ Standard Hepatic Arterial Aanatomy:

- CHA arise from celiac trunk
- CHA gives GDA and continues as HA proper
- HA proper gives RHA and LHA
- RHA gives anterior and posterior branches.
  - Anterior division of RHA divides into Sg 5 and 8 branches
  - Posterior division of RHA divides into Sg 6 and 7 branches
- LHA gives medial and lateral branches
  - Medial division of LHA divides into Sg 4A and 4B branches
  - Lateral division of LHA divides into Sg 2 and 3 branches



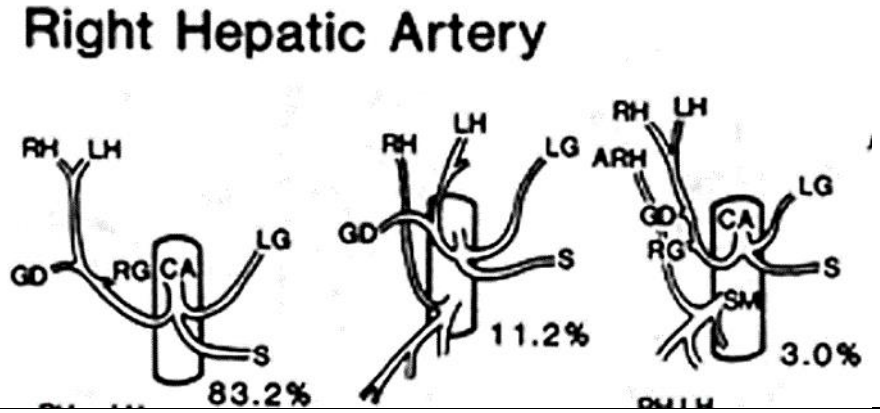




❖Variants of Hepatic Arterial Aanatomy:

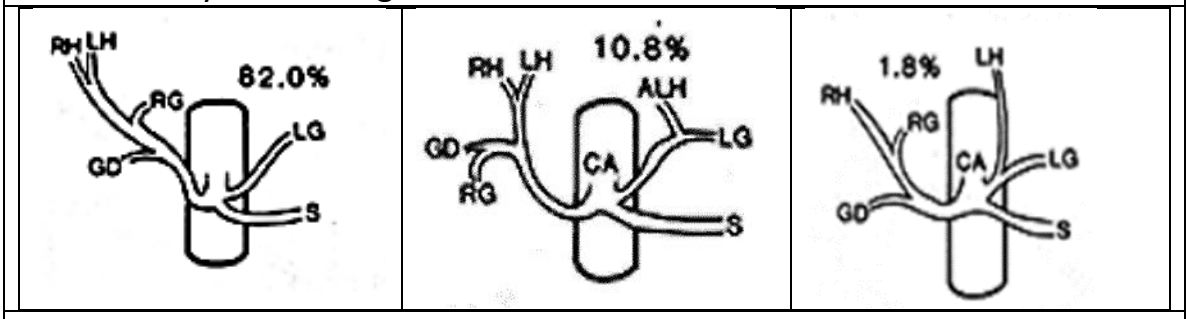
RHA:

- RHA arising directly from celiac trunk
- Replaced RHA arising from SMA
- Accessory RHA arising from SMA



LHA:

- Replaced LHA arising from LGA
- Accessory LHA arising from LGA



RHA and LHA:

- Replaced / Accesory RHA and Replaced / Accesory LHA

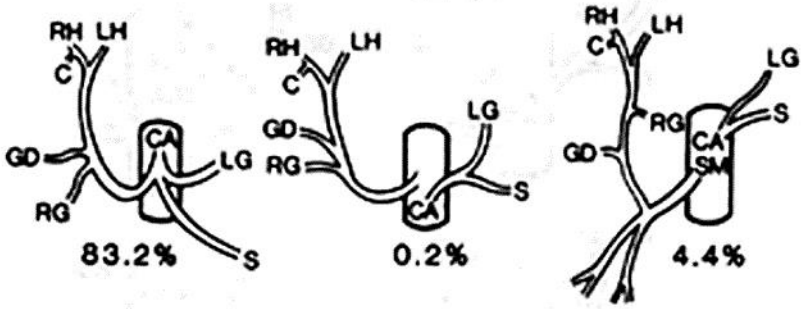
MHA:

- MHA arising from RHA or LHA
- MHA, RHA, LHA arise from HA proper (Trifurcation)

CHA:

- CHA arsing for SMA
- CHA arising for LGA
- CHA arsing directly from aorta
- CHA trifurcation into RHA, LHA and GDA

Common Hepatic Artery



Celiac Trunk:

- Common orignin of celiac axias and SMA
- Celiac axis arising from SMA

Extra-hepatic Supply to HCC:

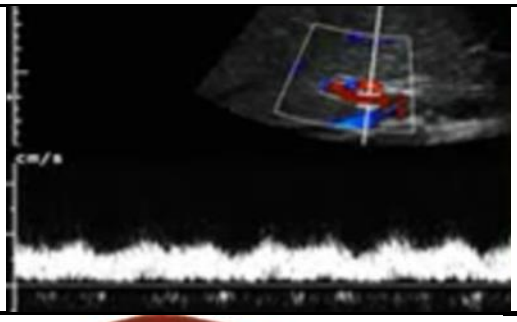
- Right and left inferior phrenic arteries (most common)
- Right and left gastric arteries
- Pancreaticoduodenal arcade
- Right and left internal mammary arteries
- Superior epigastric artery
- Intercostal artery
- Lumbar artery
- Adrenal arteries
- Capsular branches of the right renal artery
- Branches of the middle or right colic artery, and omental branches

B- Portal Venous Anatomy and Variants

- The portal vein represents a confluence of the superior mesenteric vein (SMV) and the main splenic vein

Normal Doppler Waveform:

- Continuous hepatopetal flow
- Respiratory phasacity
- Low velocity = 15 – 28 cm/sec



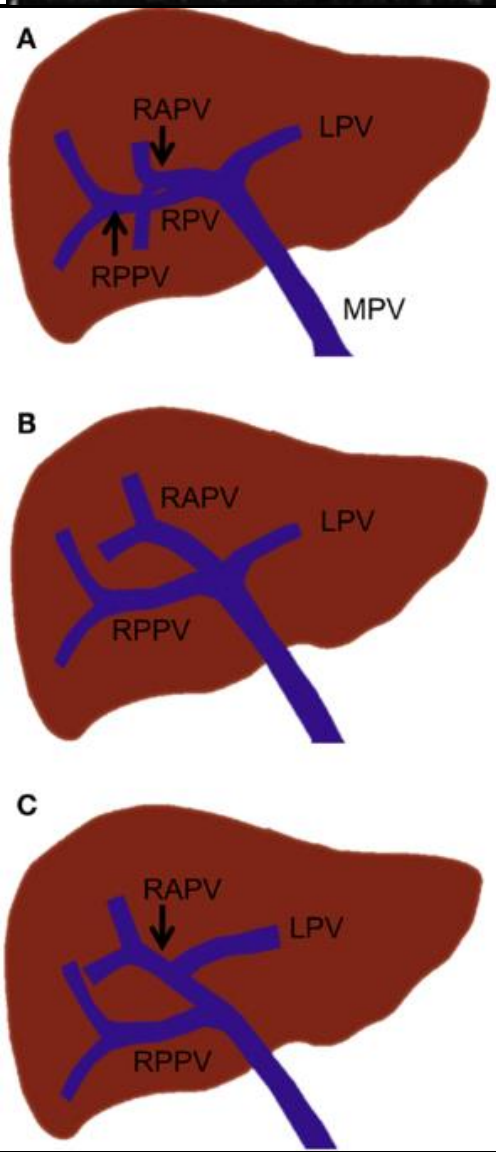
Standard Portal Vein Anatomy:

- Main portal vein divides at hepatic hilum into RPV and LPV
- LPV gives off the branches supplying Segment 2, 3 and 4
- RPV gives rise to:
  - RAPV divides into Sg 5 and 8 branches
  - RPPV divides into Sg 6 and 7 branches

Trifurcation of main portal vein:

- Main portal vein divides into RAPV, RPPV and LPV

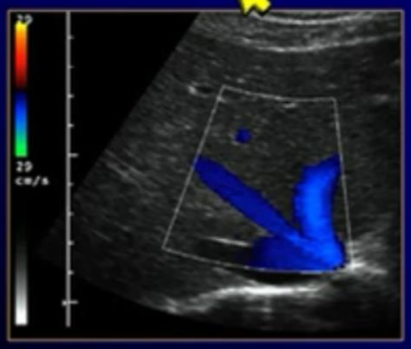
RAPV arising from LPV

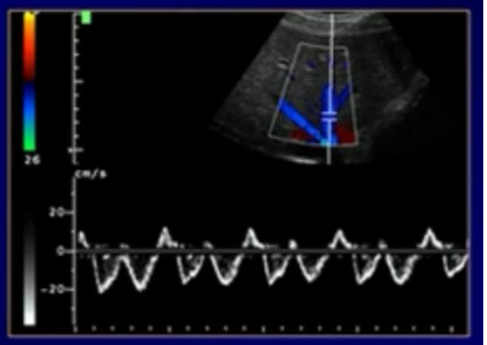


C- Hepatic Venous Anatomy and Variants

Hepatic Veins: Normal Color Doppler

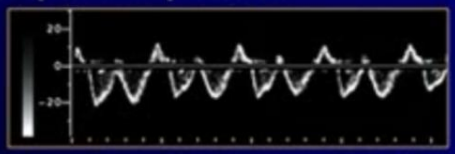
- Toward IVC and heart
- Away from transducer
- Characteristic pulsatile flow

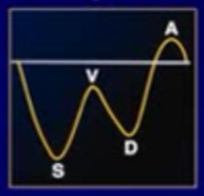




Hepatic Veins: Normal Doppler

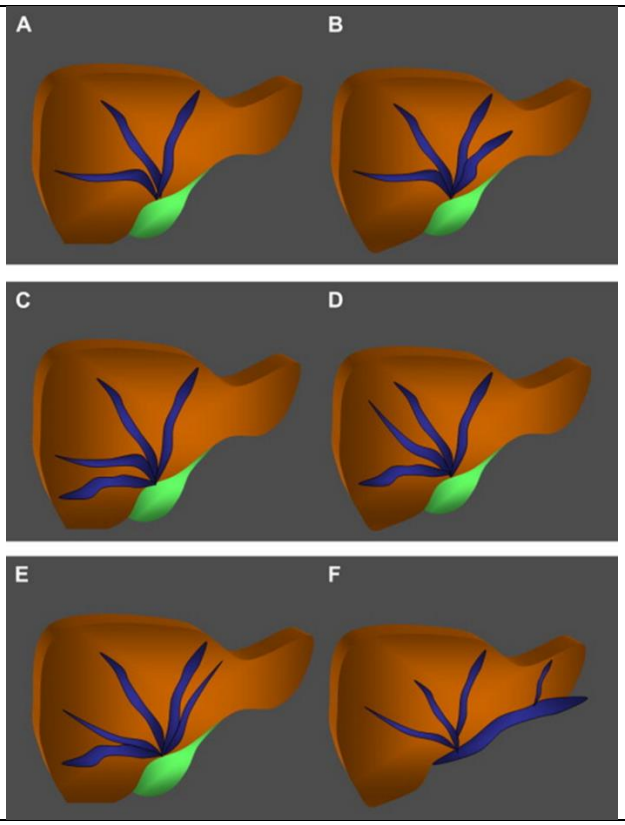
- Reflects respiratory phases
- Reflects variations in central venous pressure transmitted from the RA
- Reflects compliance of liver parenchyma
- Triphasic pattern



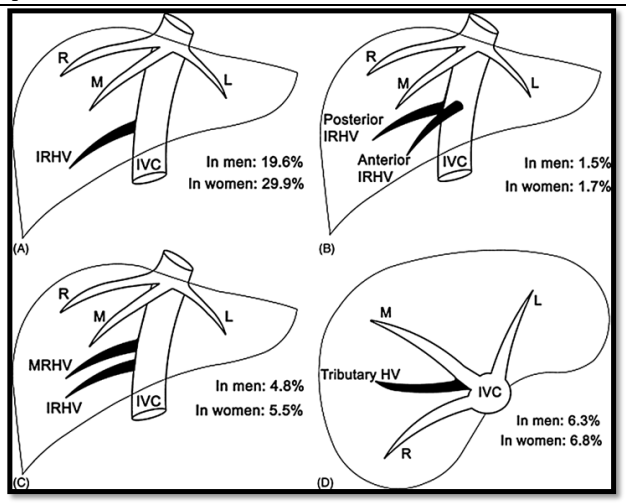


- Deep inspiration or Valsalva may reduce pulsatility of waveform in normals

A. The middle and left hepatic veins form a common trunk that drains into the vena cava.
B. Two left hepatic veins.
C. Two right hepatic veins.
D. Two middle hepatic veins.
E. Two right hepatic veins + Two left hepatic veins.
F. Accessory hepatic vein.



Accessory Inferior Right hepatic vein



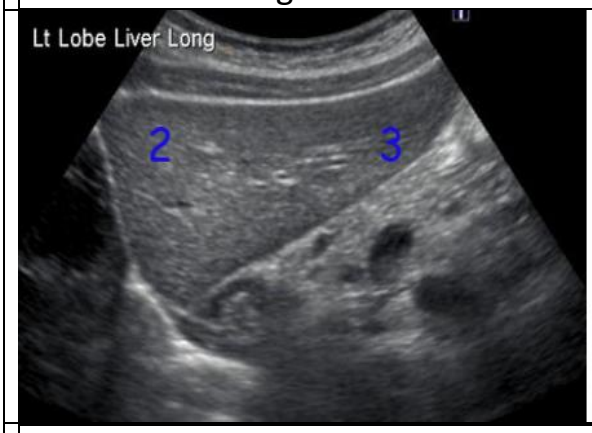
Liver Imaging Techniques

I- Liver US:

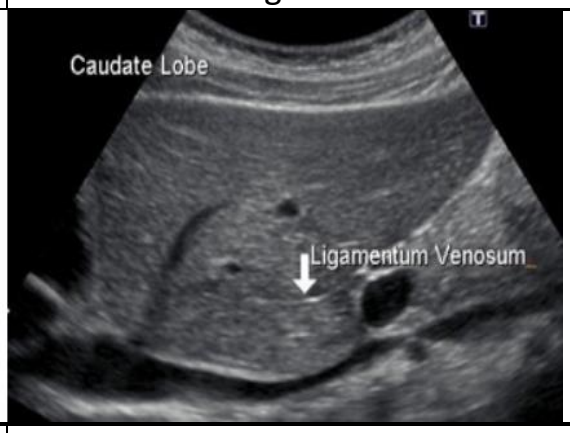
- Average size
- Homogenous parenchymal echotexture of liver > Kidney
- Hepatic veins: anechoic with no echogenic walls
- Portal veins: anechoic with echogenic walls
- Diaphragm and capsule: Thin uniform echogenic line

A) Longitudinal Sections (from Left to Right):

1) Lt Parasagittal:
   Lt lobe Seg 2and 3



2) Mid-sagittal:
   Lt lobe Seg IV

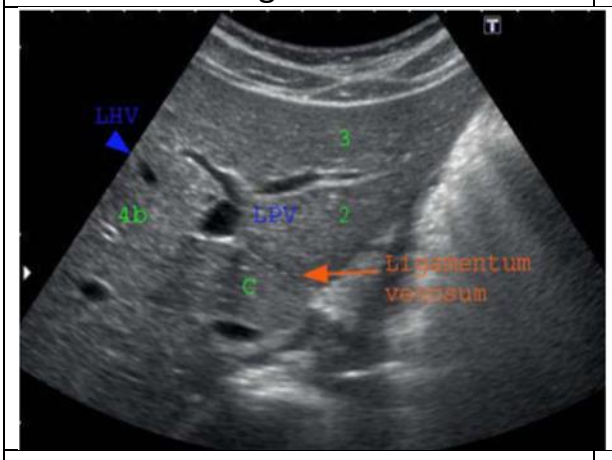


3) Rt Para-sagittal:
   Rt Lobe seg 8 and 5

4) Rt axillary:
   Ri Lobe seg 6 and 7

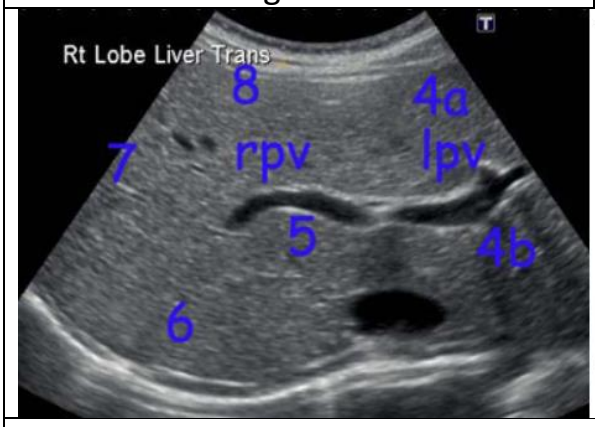
B) Transverse Sections

1) Lt Transverse Inferior:
Lt lobe seg 3 and 4b



2) Lt Transverse Superior:
Lt lobe seg 2 and 4A

3) Rt Transverse Mid:
Rt lobe seg 5 and 6



4) Rt Transverse Superior:
Rt lobe seg 7 and 8

C) Oblique Sections

Porta hepatis

Oblique angle 45degree rotation from the sagittal view to the transverse view

Causes of Generalized Reduction of Liver Echogenicity:

- Acute hepatitis
- Diffuse malignant infiltration

Causes of Generalized Increased Liver Echogenicity:

- Diffuse fatty infiltration
- Cirrhosis: can be coarsened as well
- Chronic hepatitis 3: can be coarsened as well
- Diffuse infiltration or deposition
- Malignant process
- Granulomata
    - Tuberculosis
    - Brucellosis
    - Sarcoidosis
- Glycogen storage disease
- Haemochromatosis

Causes of Coarsened Hepatic Echotexture

- Hepatic fibrosis
- Cirrhosis
- Diffusely infiltrating hepatic metastases
- Hepatic lymphoproliferative diseases
    - Hepatic lymphoma
    - Hepatic leukaemia
- Metabolic abnormalities
    - Hepatic amyloidosis
    - Glycogen storage disease

II- Triphasic CT

Liver Dual Blood Supply:

- Hepatic artery (25% of vascularization)
- Portal vein (75% of vascularization)

Normal liver parenchyma:

- Maximum enhancement in the hepatic phase (portal venous phase) at 70-80 sec post injection
- Minimal enhancement in the late arterial phase at 25-35 sec post injection

Hypervascular Liver Tumors (eg: HCC, HA, FNH)

- 100% of their blood supply from the hepatic artery.
- Enhance and best seen in the late arterial phase.
- Normal parenchyma shows only minimal enhancement.

Hypovascular Liver Tumors (eg: Metastasis):

- Late arterial phase: hypovascular liver tumors enhance poorly and the surrounding liver does also enhance poorly in that phase.
- Portal venous phase (hepatic phase): hypovascular tumor is best seen when the surrounding tissue enhances.

Phases:

1) Non-enhanced CT (NECT)
2) Late arterial phase: 30-35 sec post injection
3) Portal venous phase (Hepatic phase): 60-75 sec post injection
4) Delayed phase (Equilibrium phase): 3-5 min post injection

Volume of contrast: 150cc contrast
Rate of injection: 5cc/sec. through 18 gauge green venflon

CT density:

- Homogenous CT density
- NCCT: Liver > Spleen by 10 HU

III- Dynamic MRI

Patient Position:

- Patient supine
- Head towards magnet
- Body coil
- Axial: Whole liver form diaphragm to iliac crest
- Coronal: Whole liver from anterior to posterior abdominal wall
- FOV = 350
- Slice thickness = 3mm

- Protocol:
- Axial T1 in-of-phase
- Axial T1 out-of-phase
- Axial T2 SSFSE
- Axial T2 FS
- Axial DWI
- Axial ADC
- Coronal T2
- Axial Dynamic 3D (5phases) (VIBE or LAVA)
    1) Pre-contrast phase
    2) Early arterial phase: 20-25 sec post injection
    3) Late arterial phase: 30-35 sec post injection
    4) Portal venous phase (Hepatic phase): 60-75 sec post injection
    5) Delayed phase (Equilibrium phase): 3-5 min post injection
- Axial and coronal Delayed 2D

MRI signal pattern:

- Homogenous intermediate intensity.
- T1: SI of liver (= pancreas) > spleen/kidney.
- T2: SI of liver < spleen/Kidney

❖MRI Hepatic Specific Contrast Agents:

5 types are currently used:

1) **Non-Specific (ECF) Gadolinium Agents**
2) **Hepatobiliary Specific Contrast Agents**
3) **Combined Gadolinium and Hepatobiliary-Specific Contrast Agents**
4) **RES-Specific Contrast Agents**
5) **Dual RES and Gadolinium Contrast Agents**

1) Non-Specific (ECF) Gadolinium Agents

Mechanism of Action

- Circulates and then freely distributes in the extracellular fluid compartment

Elimination

- Renal

Dosage and administration

0.1 mmol/kg 2-4 ml/sec. 20-30ml saline flush

Dynamic: 5 phases

2) Hepatobiliary Specific Contrast Agents

- Manganese is chelated to dipyridoxyl diphosphate to produce agent known as mangafodipir trisodium (**Teslascan, Mn-DPDP**)

Mechanism of Action

- Mn => Paramagnetic
- Dipyridoxyl Diphosphate => Uptake by hepatocytes (B6)

Dosage and administration

- 0.5 mL/kg slow infusion over 10–20 minutes
- No dynamic study
- Timing of acquired delayed: 10-30 min

Elimination

- Biliary (59%) 5 Days
- Renal (15 %)

Indications for Use

- Differentiate lesions of hepatocellular origin from lesions of non-hepatocellular origin
- Surveillance of the liver for metastases
- Contrast- enhanced MRCP

3) Combined Gadolinium and Hepatobiliary-Specific Contrast Agents

2 agents are currently available

- Gadobenate Dimeglumine (MultiHance, Gd-BOPTA)
- Gadoxetic acid (Primovist, Gd-EOB-DTPA)

Elimination

- Gd-BOPTA 3%–5% biliary, 95% renal
- Gd-EOB-DTPA 50% biliary, 50% renal

Dosage and administration

- Gd-BOPTA 0.1 mmol/kg (2 mL/sec)
- Gd-EOB-DTPA 0.025 mmol/kg (2 mL/sec)

Dynamic phases + Delayed hepatobiliary phase

- Gd-BOPTA 1–2 hours
- Gd-EOB-DTPA 10 minutes to hours

Indications for Use

- Dynamic phase imaging is particularly important for lesion characterization
- Delayed phase imaging in the hepatobiliary phase increases the sensitivity for MRI for liver lesion detection
- Differentiate lesions of hepatocellular origin from lesions of non-hepatocellular origin
- Focal nodular hyperplasia can be accurately differentiated from hepatic adenoma, thereby eliminating the need for lesion biopsy

4) RES-SPECIFIC CONTRAST AGENTS

Two agents are currently available

- Super paramagnetic iron oxide particles (SPIO, Ferumoxides (Feridex and Endorem) mean particle size > 50 nm)
- Ultra-small super paramagnetic iron oxides (USPIO (Resovist, SH U 555 A) mean particle size < 50 nm).

Mechanism of Action

- Particles are phagocytosed by macrophages throughout the body but are preferentially entrapped by Kupffer cells, which line the hepatic sinusoids
- Act as a negative contrast agent: Reduced signal intensity on T2 and T2*
- Lesions that contain no Kupffer cells remain unchanged, while the signal intensity of the normal liver is reduced on T2 WI

Indications for Use

- Detection of HCC and metastasis in high risk patient
- Possible characterization of FNH
- MR angiography (USPIO)

Delayed (accumulation) phase:

SPIOI: 1–4 hours after infusion (T2- and T2* WI)
USPIO: 10 min and 8 hrs (T2- and T2* WI)

5) Dual RES Specific and Gadolinium Contrast MRI

Advantages

- On dynamic T1-weighted images the background liver is darkened by the SPIO particles while the lesion of interest is lightened by gadolinium
- Improve lesion-to-liver contrast
- SPIO improve the visibility of hypovascular HCC that would be missed at imaging with gadolinium alone, and the gadolinium allows detection of hypervascular, well-differentiated HCC that would be missed at imaging with the use of SPIO particles alone.

Imaging protocol

- Infusion of SPIO over 30 minutes, then wait 30–60 minutes.
- Apply axial T2 &T2*-WI.
- Administer gadolinium injection.
- Apply the axial T1-wi during the late hepatic arterial phase, the portal venous phase, and the equilibrium phase of enhancement.

I- Diffuse Liver Diseases

❖ **Inflammatory Diseases** ○ **Viral hepatitis** ○ **Fatty liver** ○ **Cirrhosis and fibrosis** ○ **Hepatic Schistosomiasis** ○ **Hepatic Sarcoidosis** ○ **Hepatic Amyloidosis**
❖ **Metabolic Diseases:** ○ **Hemochromatosis** ○ **Wilson disease** ○ **Glycogen Storage Diseases** ○ **Lipid Storage Diseases** ○ **Mucopolysaccharidoses**
❖ **Hematological Diseases**
❖ **Vascular Diseases:** ○ **Portal Venous Thrombosis and Cavernous Transformation** ○ **Budd-Chiari Syndrome.** ○ **HELLP Syndrome** ○ **Hepatic Arterial Obstruction** ○ **Passive hepatic congestion**
❖ **Neoplastic:** ○ **Lymphoma** ○ **Leukemia**

A- Acute Hepatitis

- Liver suffers an injury with a resulting inflammatory reaction.

Aetiology:

- Viral hepatitis (e.g. HAV, HCV, HBV)
  - Acute viral hepatitis <6m
  - Chronic viral hepatitis >6m
- Parasites
- Drug-induced/toxin-induced hepatitis
  - e.g. Acetaminophen/paracetamol toxicity
- Alcoholic hepatitis
- Steatohepatitis

Clinical Picture:

- Fever, abdominal pain, and jaundice.
- Elevated liver functions: AST, ALT, and GGT.
- Specific serum immunologic markers may be present (e.g. anti-HAV IgM indicates active hepatitis A infection).

❖ Imaging Findings of Acute Viral / Alcoholic Hepatitis

US:

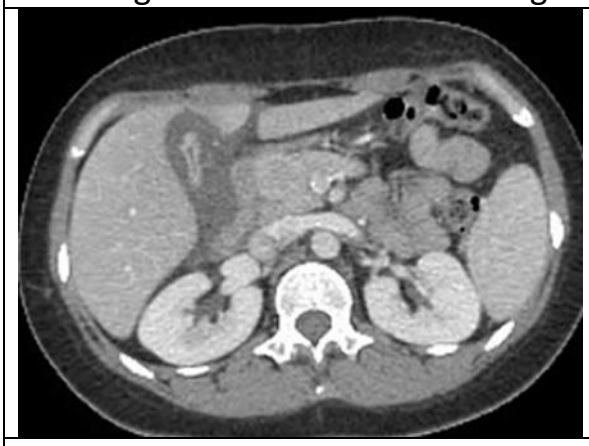
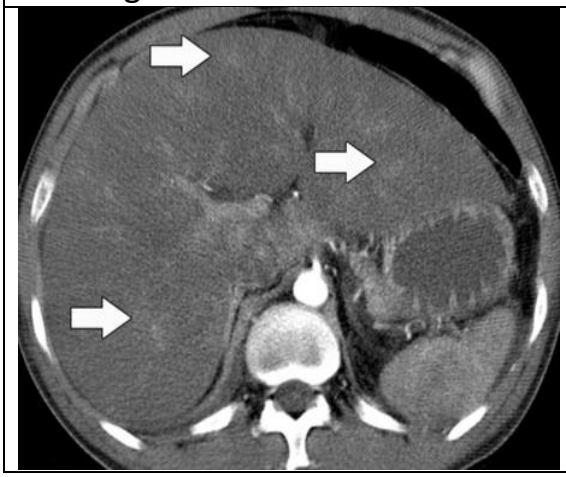
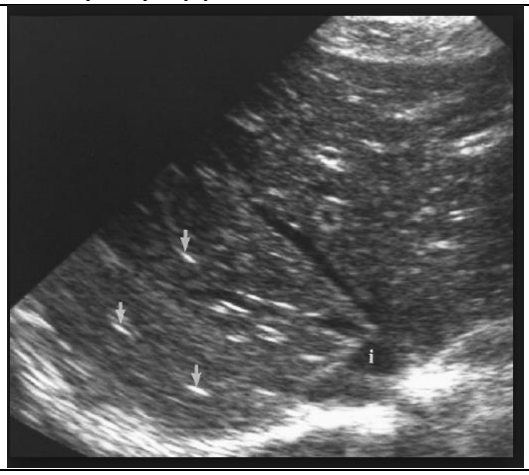
- Hepatomegaly (most sensitive sign) >15.5 cm at the midclavicular line
- Diffuse decreased hepatic echotexture
- Diffuse gallbladder wall thickening
- "Starry sky appearance" = ↑ Echogenicity of portal venous walls
- Periportal oedema
- Doppler => Normal

CT (Not first line imaging modality for evaluation)

- Hepatomegaly >15.5 cm at the midclavicular line
- NCCT: Diffuse decreased parenchymal attenuation
- CECT: Diffuse heterogeneous parenchymal enhancement
- Diffuse gall bladder wall thickening
- Decreased attenuation around the portal system and at the hepatic hilum (periportal oedema)
- ± Periportal/hepatoduodenal lymphadenopathy

MRI:

- T2:
  - Increased T2 signal around the portal system (periportal oedema)
  - Mild diffuse increase in parenchymal signal intensity
- T1 C+ (Gd):
  - Delayed, gradual periportal enhancement

Diffuse gall bladder wall thickening	Periportal hypodensity
	
Heterogeneous enhancement	**Starry sky appearance**
	

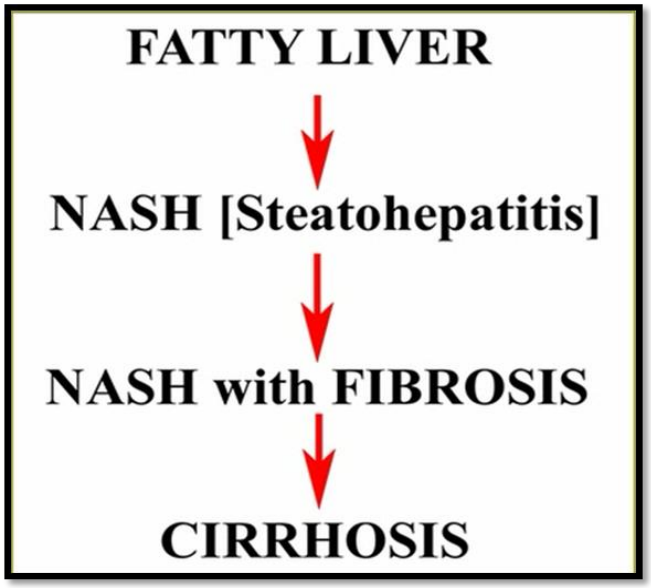
B- Fatty Liver

- Fatty liver disease is the most common cause of chronic liver disease.
- Hepatic steatosis is characterized by increased accumulation of triglyceride content within hepatocytes of liver.

Risk factors associated with diffuse steatosis including:

- Obesity
- Malnutrition
- Diabetes mellitus
- Steroid use
- Alcoholic liver disease
- Pregnancy
- Hepatitis

Nonalcoholic Fatty Liver Disease (NAFLD):



Liver Biopsy:

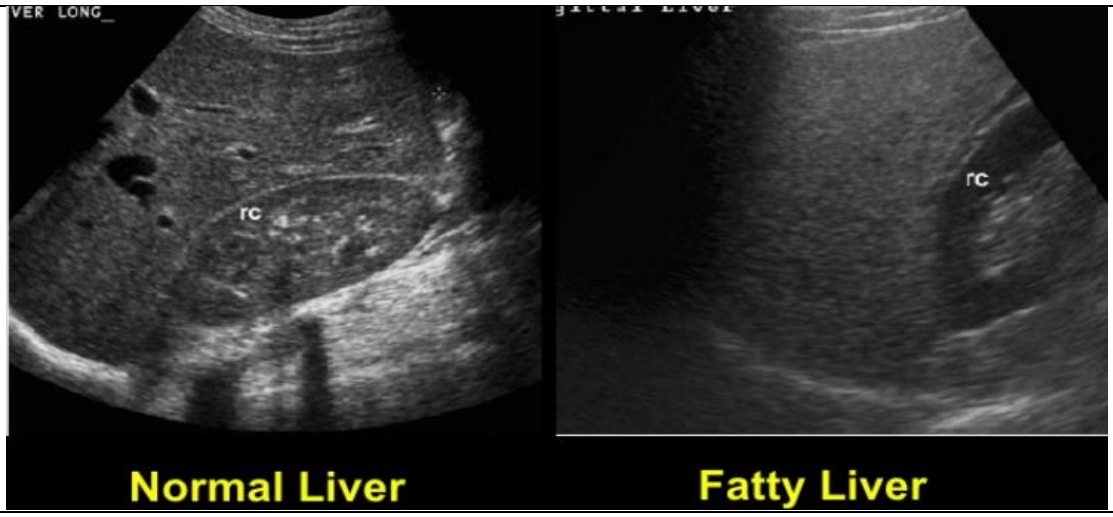
- Histopathological evaluation is the most accurate method for the diagnosis of NASH, which is based on the assessment of:
  a) **Macrovesicular steatosis**
  b) **Necroinflammatory activity**
  c) **Fibrosis.**

Imaging findings:

- Diffuse fatty infiltration
- Focal nodular fatty infiltration
- Lobar, segmental (Wedge shaped)

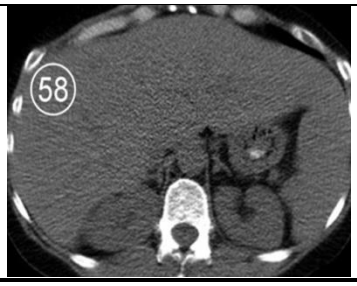
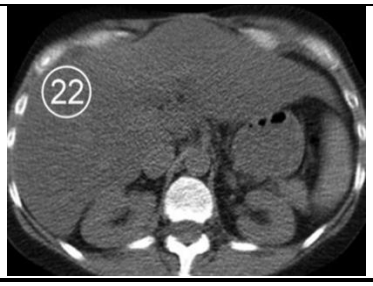
US:

- Mild steatosis: Liver echogenicity > Right kidney echogenicity.
- Moderate steatosis: Liver echogenicity obscures visualization of hepatic and portal vein wall.
- Marked steatosis: Posterior attenuation avoids deep liver parenchyma evaluation.



NCCT:

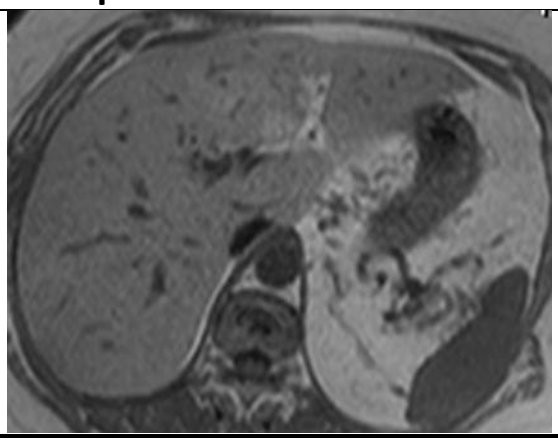
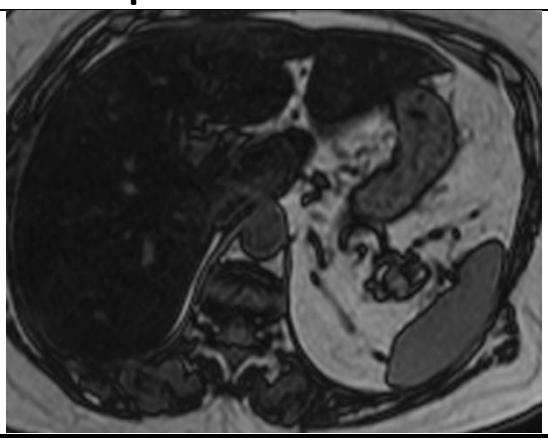
- Normal: Liver attenuation > Spleen attenuation by 8–10 HU
- Increased diffuse deposition of fat leads to a decrease in attenuation.
- Mild steatosis: Hepatic vessels < hepatic parenchyma
- Moderate steatosis: Hepatic vessels = hepatic parenchyma
- Marked steatosis: Hepatic vessels > hepatic parenchyma

Mild staetosis	Moderate steatosis	Marked steatosis
		

MRI

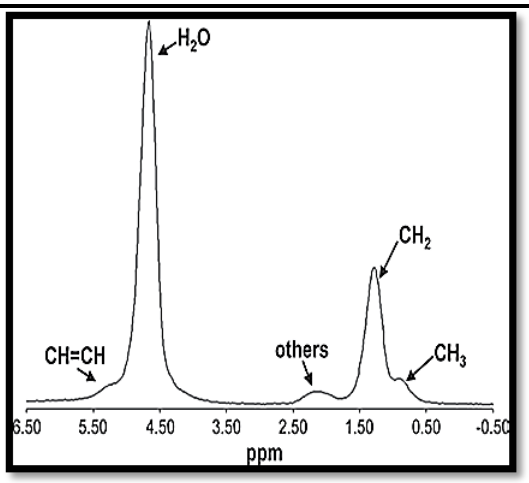
A) Chemical shift imaging:

- T1WI in-phase gradient echo image
    - Increased signal intensity of fatty liver than spleen
- T1WI out-of-phase gradient echo image
    - Signal drop out= Decrease or loss of signal intensity of fatty liver

In of phase	Out of phase
	

B) MR Spectroscopy:

- **The lipid (triglyceride) spectrum consists of multiple peaks:**
    - CH3 (0.9 ppm)
    - CH2 (1.3 ppm)
    - CH2 (2.0 and 2.2 ppm)
    - CH (5.3 ppm)



❖Patterns Of Focal Fatty Infiltration And Sparing

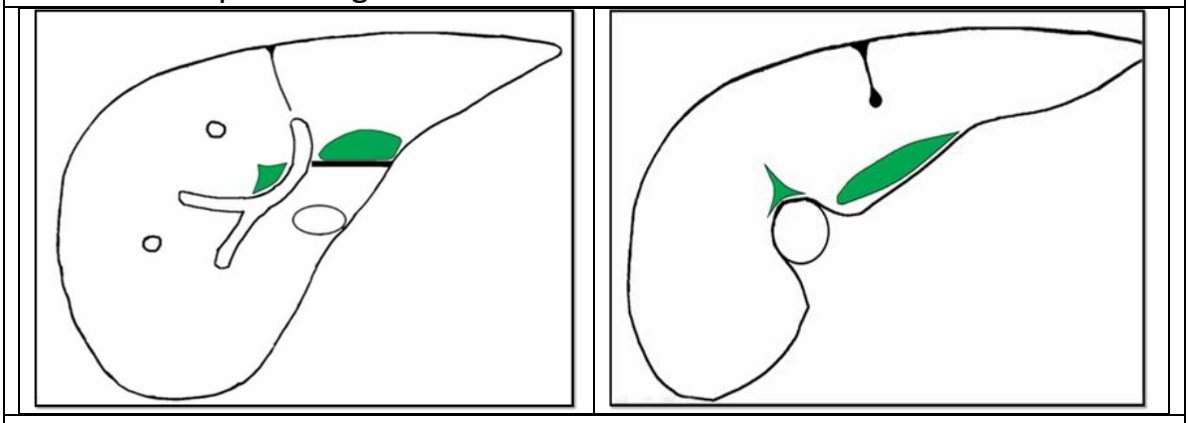
1) Focal Fatty Infiltration

Imaging findings:

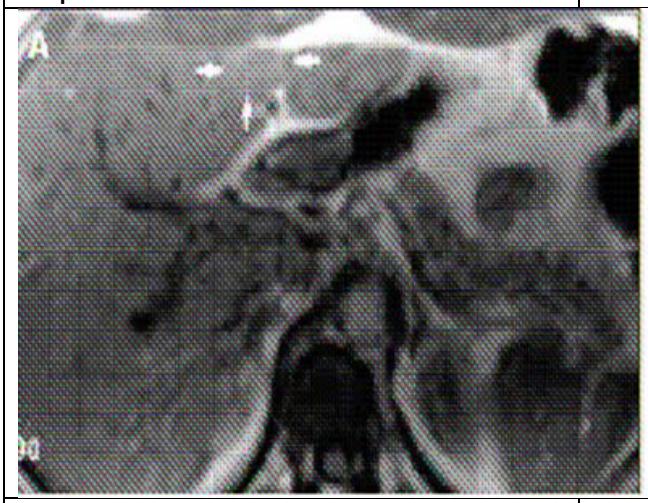
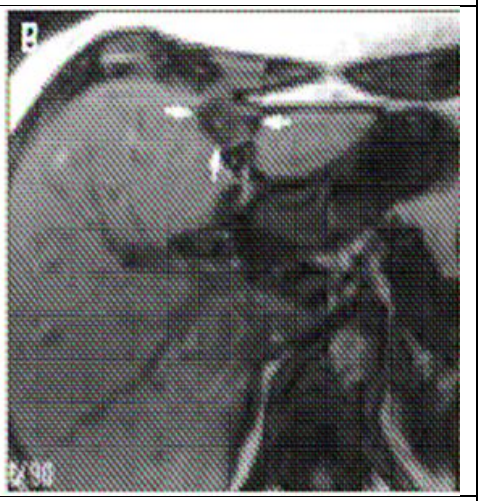
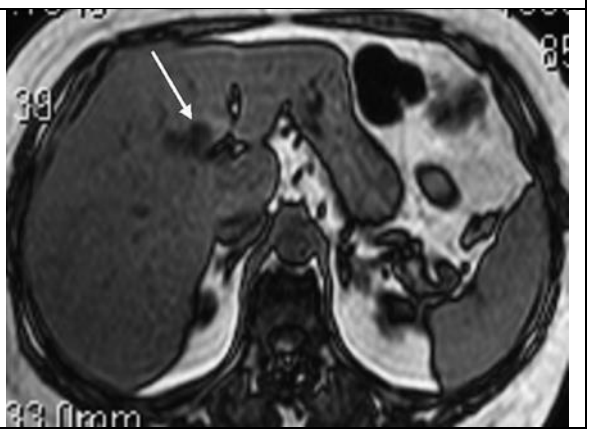
- Geographic but may be nodular.
- No mass effect
- Coursing of normal vascular structures through the fatty area
- Enhancement on CT and MRI indistinguishable from that of normal liver.

Location:

- The medial aspect of the left lobe
- Along the falciform ligament
- Gallbladder fossa
- Central aspect of segment IV
- Adjacent to the porta hepatis.
- The subcapsular region



- US: Focal well-circumscribed, echogenic area.
- CT: Focal areas of relative low attenuation.
- In-phase T1 GRE: focal high signal
- Out-phase t1 GRE: signal drop out

In-phase	Out-phase
	
	

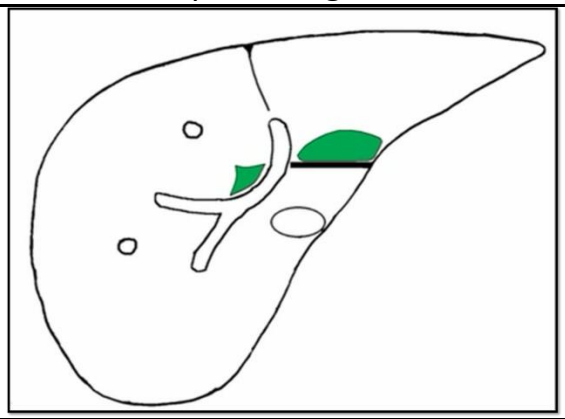
2) Focal Fatty Sparring

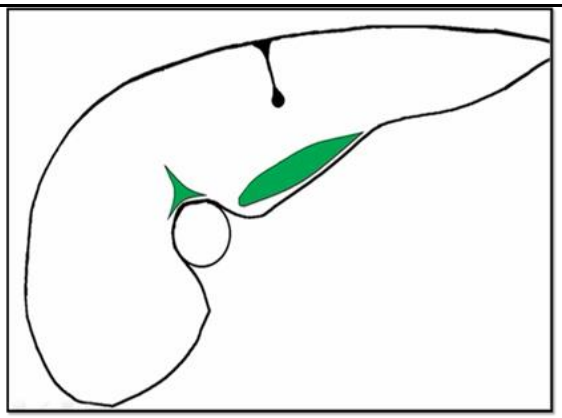
Imaging findings:

- Geographic but may be nodular.
- No mass effect
- Coursing of normal vascular structures through the fatty area
- Enhancement on CT and MRI indistinguishable from that of normal liver.

Location:

- The medial aspect of the left lobe
- Along the falciform ligament
- Gallbladder fossa
- Central aspect of segment IV
- Adjacent to the porta hepatis.
- The subcapsular region





Imaging findings:

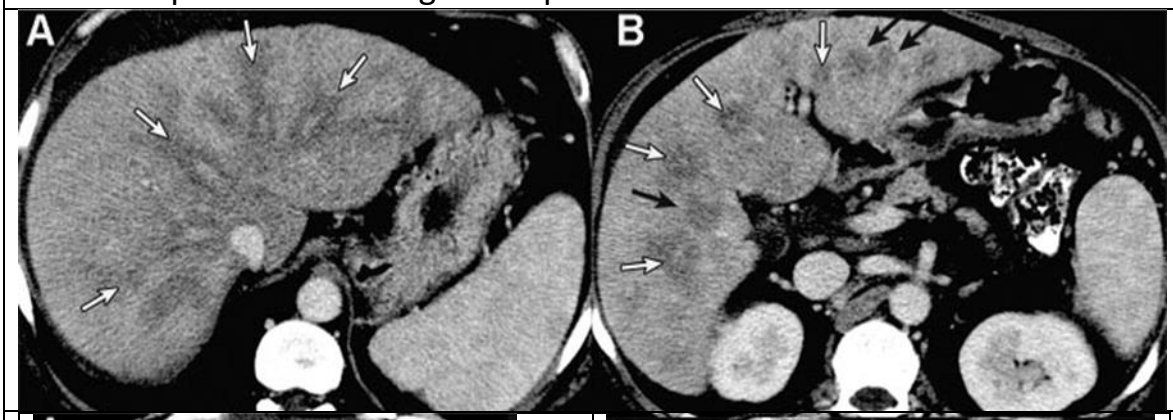
- US: focal hypoechoic lesion in echogenic steatotic liver.
- CT: focal hyperdense area within diffusely hypodense liver parenchyma.
- Out-phase T1 GRE: focal fat sparring lack of signal drop and appear hyperintense relative to surrounding steatosis with signal drop

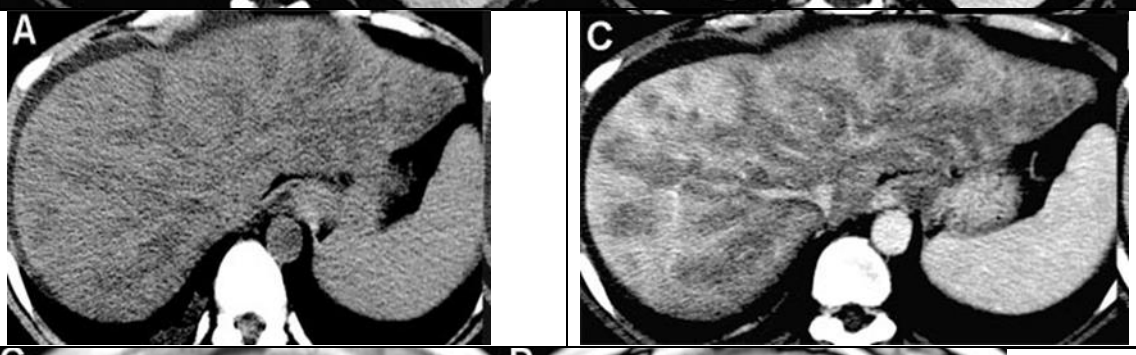
3) Perivascular Steatosis

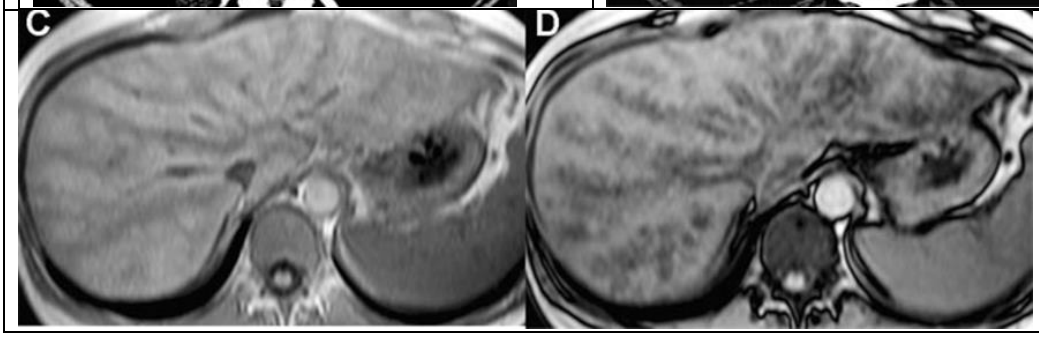
- Periportal fatty infiltration: around portal veins
- Perivenular fatty infiltration: around hepatic veins
- Combined: it surrounded both the hepatic vein and the portal tract.

Imaging findings:

- Glove like, parallel, uniformly thin (5–15mm) halos around the vessels.
- Depending on vessel orientation, halos were:
    - Tram like (vessel segments parallel to scanning plane)
    - Round (vessel segments perpendicular to scanning plane)
- CT: hypodense areas along the portal and/or hepatic veins
- In-phase T1 GRE: high signal
- Out of phase T1 GRE: signal drop







4) Intratumoral Steatosis

Fat within the lesions can be in the form of:

- Macroscopic lipid
- Intracellular lipid

<u>These lesions are:</u>

- **Hepatic adenoma**
- **Hepatocellular carcinoma**
- **Regenerative nodules**

5) Perilesional Steatosis

- US: It appears as bright echogenic rim.
- Out of phase T1 GRE: hypointense rim around metastatic lesions.

6) Subcapsular Staetosis

- In diabetic patients receiving insulin through peritoneal dialysis, characteristic subcapsular steatosis can be observed due to local action of insulin at the surface of the liver

C- Hepatic Fibrosis

- Liver fibrosis, a common feature of almost all causes of chronic liver disease, involves the accumulation of collagen, proteoglycans, and other macromolecules within the extracellular matrix

Fibrosis progress leading to:

- Hepatic dysfunction
- Portal hypertension
- Cirrhosis

❖ Liver biopsy

- Is the standard of reference for diagnosing liver fibrosis
- Invasive and costly
- Subject to complications and sampling variability.

Change	Score
No fibrosis	0
Fibrous expansion of some portal areas, with or without short fibrous septa	1
Fibrous expansion of most some portal areas, with or without short fibrous septa	2
Fibrous expansion of most some portal areas with occasional bridging	4
Marked bridging with occasional nodules (incomplete cirrhosis).	5
Cirrhosis, probable or definite	6

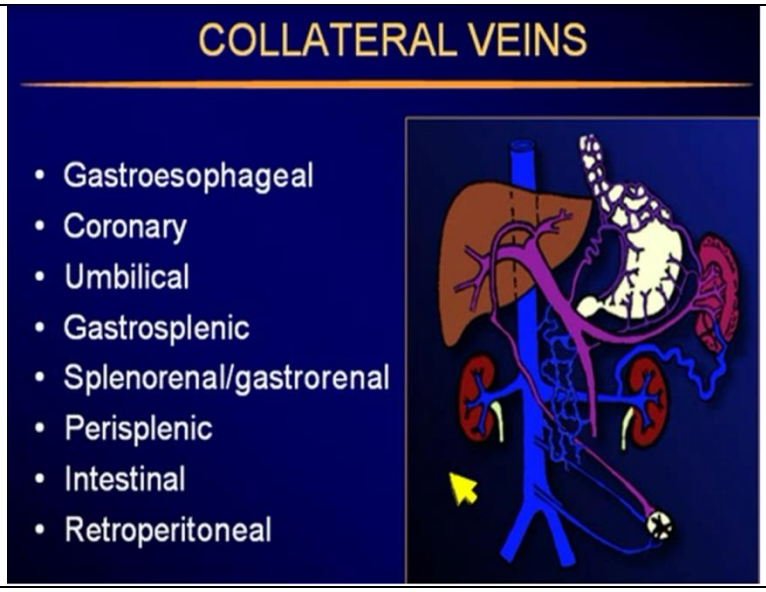
Imaging Findings:

i) Morphological Changes:

- Surface nodularity
- Widening of fissures
- Expansion of the gallbladder fossa
- Notching of the right lobe
- Atrophy of the right lobe
- Relative enlargement of the lateral segments of the left lobe and caudate lobe (C/R > 0.65)

ii) Secondary Signs of portal Hypertension:

- Splenomegaly (Length > 12 cm & Width > 6 cm) + splenic siderotic nodules (Gamna–Gandy bodies)
- Portal vein abnormalities:
    - Dilated portal vein > 13 mm
    - Dilated SMV and splenic vein > 10mm
    - Decreased portal vein velocity
    - Biphasic / Reversed portal vein flow
    - Secondary hepatic artery enlargement
- Portosystemic varices



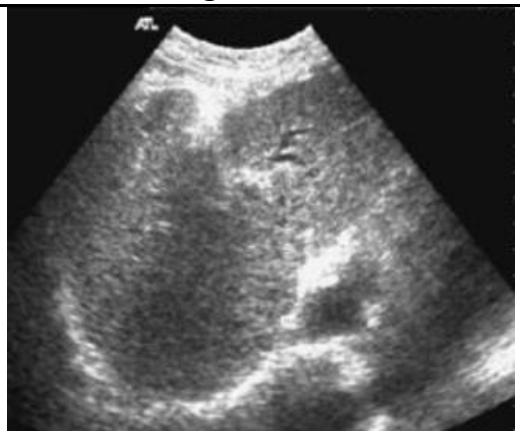
iii) Secondary Signs of Hepatic failure:

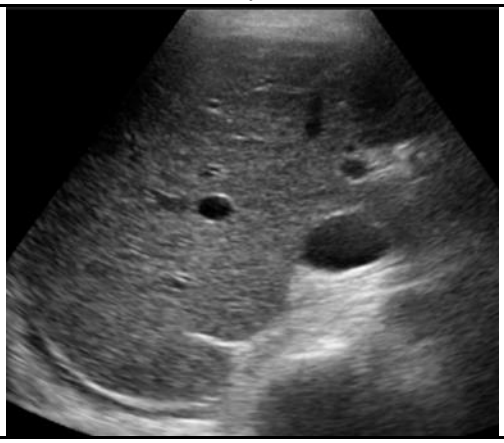
- Ascites
- Diffuse gall bladder wall thickening
- Diffuse bowel wall thickening

iv) Parenchymal Changes:

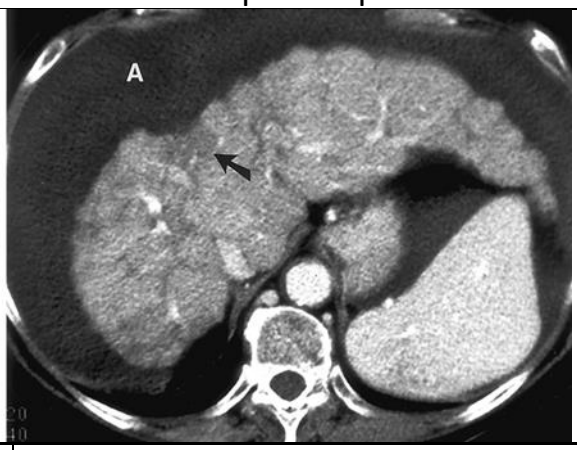
1) Conventional Imaging

- US: heterogeneous echotexture (Coarse motheaten)

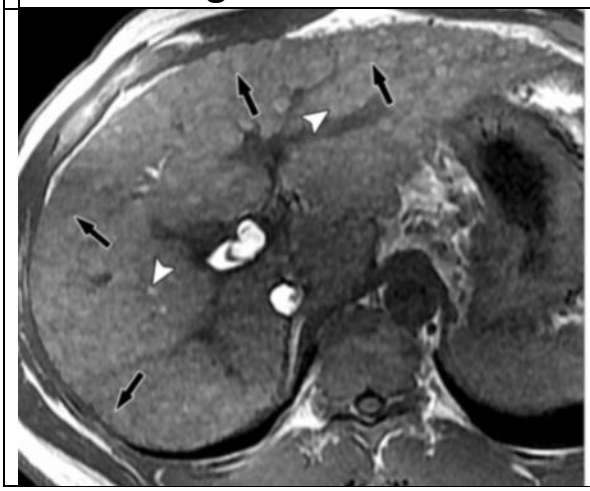




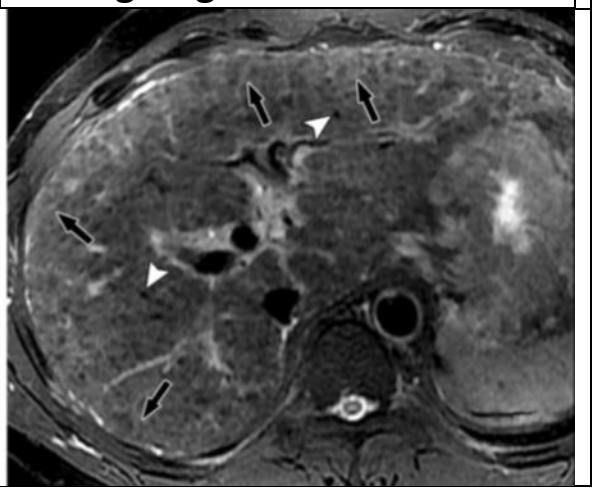
- NCCT: Hypoattenuating areas that appeared patchy or bandlike, lacelike, or confluent, accompanied by retraction of the hepatic capsule



T1: Low signal reticulations



T2: High signal reticulations



2) Sono-elastography

US-based techniques include

- Transient elastography (TE)
- Shear wave elastography (SWE)
- Acoustic radiation force impulse (ARFI) elastography
- Real-time elastography (RTE)

- Estimate parenchymal stiffness and, indirectly, the stage of fibrosis

Qualitative assessment:

- Provides a color-coded representation of the relative elasticity of tissues included in the B-mode image

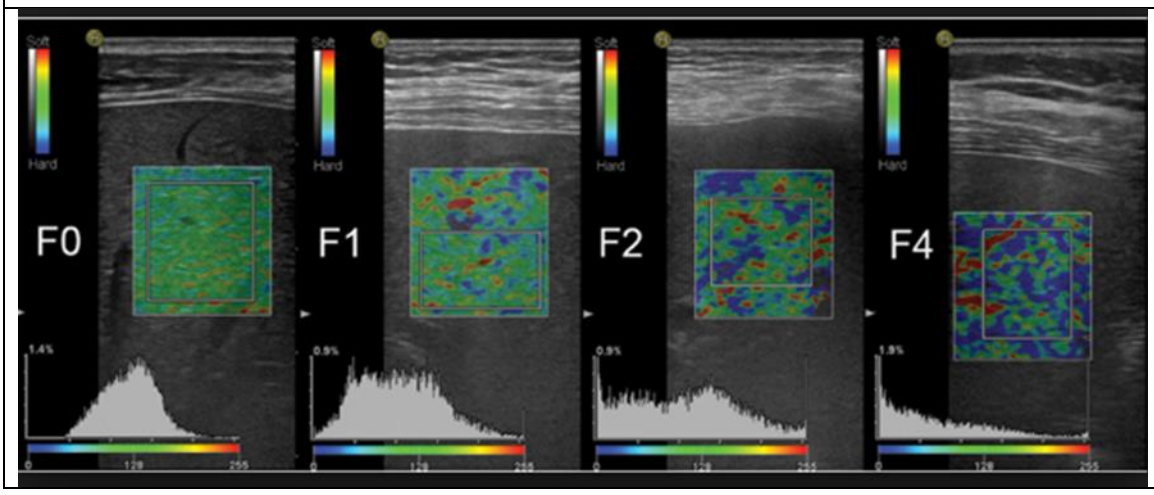
Semi-quantitative methods:

a) **Histogram of pixel distribution:**
   - Each pixel of the image has a specific numeric value to represent tissue elasticity

b) **Elastic ratio**
   - is the ratio of strain distribution in two selected ROIs

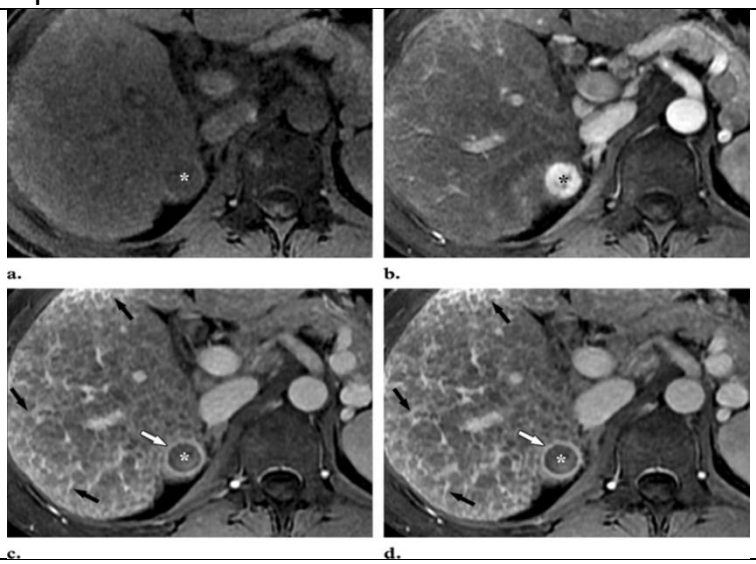
c) **Fibrosis index (Elastic index)**

- A pulse-echo US acquisition is then used to detect the wave propagation velocity, which increases proportionally with tissue stiffness.
- >7 kPa: advanced fibrosis
- 12.5-15 kPa: Cirrhosis



3) Dynamic MRI:

- Fibrotic reticulations and fibrous capsule enhance progressively after contrast agent administration and peaks during the late venous and equilibrium phases

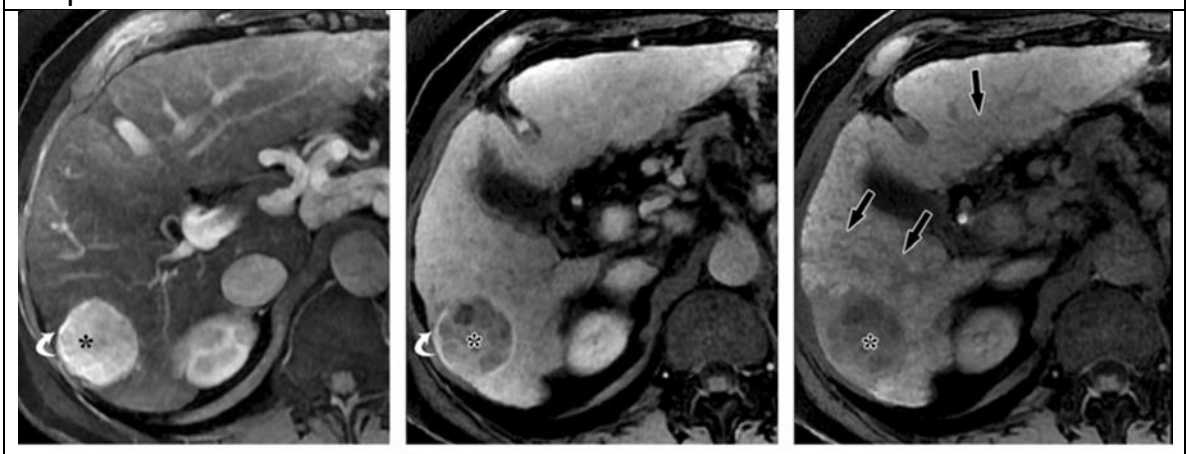


<u>Confluent Fibrosis (DD: HCC)</u>

- A wedgelike or geographic shape with straight or concave borders, radiates from the portal hilum to contact the liver surface
- Volume loss with capsular retraction
- Similar signal intensity and enhancement features as fibrotic septa
- Persistence of enhancement into the late phases

4) Hepato-billiary Gd Enhanced Dynamic MRI

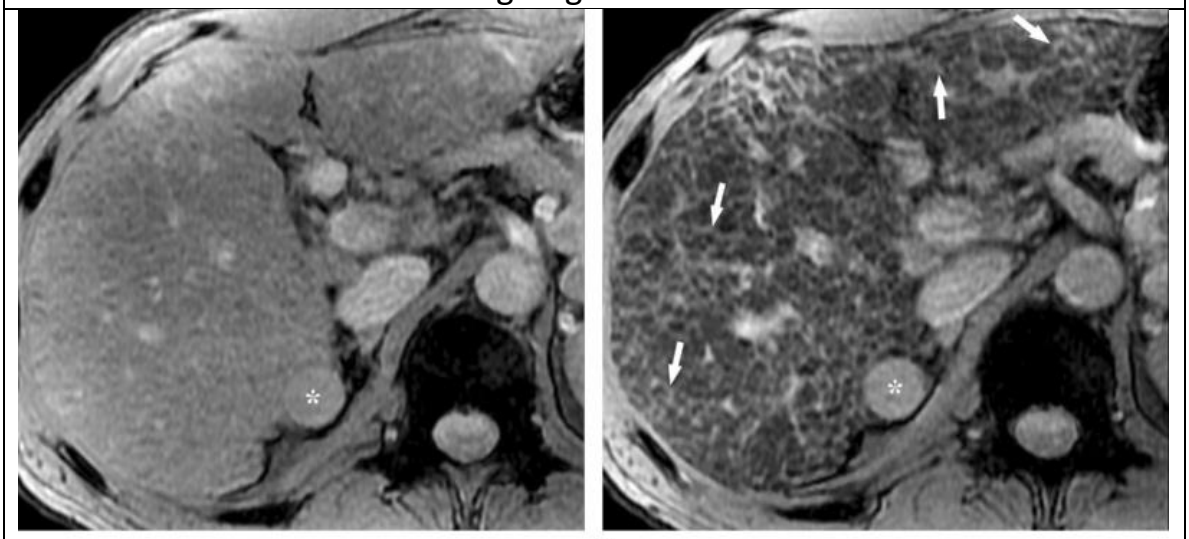
- Fibrotic reticulations appear of low signal intensity on hepatocellular phase



a. b. c.

5) SPIO (Superparamagnetic Iron Oxide) Enhanced MRI:

- Normal liver parenchyma => Kupffer cells => accumulate iron oxide => Low signal on T2*.
- Fibrotic reticulations => reduced Kupffer cell density => don't accumulate iron oxide => High signal on T2*



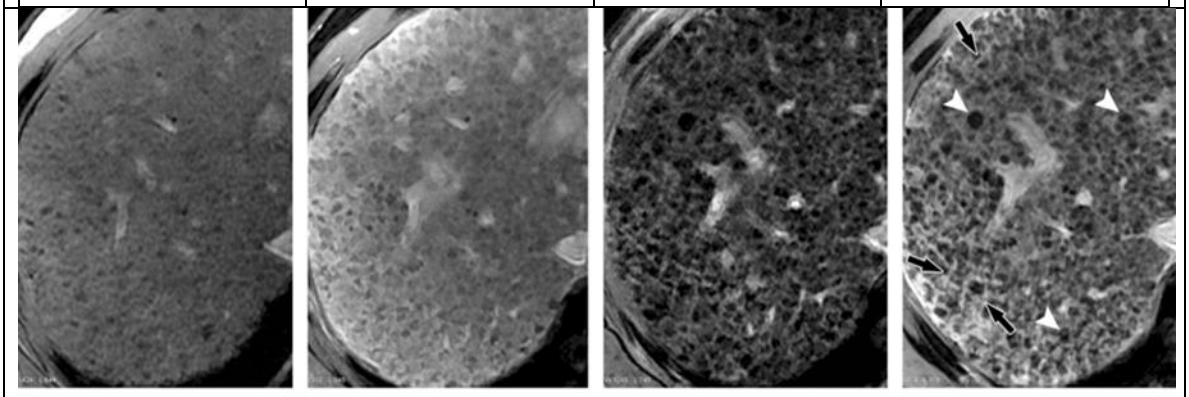
a. b.

6) MRI Double Contrast (Gd+SPIO):

- MRI double enhanced with superparamagnetic iron oxide and gadolinium in the diagnosis of advanced liver fibrosis.

- SPIO particles accumulate in hepatic Kupffer cells, causing signal loss in liver parenchyma on T2*.
- Gadolinium-based contrast agent causes delayed enhancement of hepatic septal and bridging fibrosis.
- => High image contrast between the low SI liver parenchyma and high SI fibrotic reticulations

Axial T1	Gd enhanced	SPIO enhanced	Gd+SPIO



7) DWI & ADC

- ADC of cirrhotic liver is lower than that of normal liver.
- Increased hepatic steatosis and collagen deposition in fibrosis and cirrhosis are associated with a reduction in ADC.
- ADC cutoff for fibrosis < 1.31 X 10-3 mm2/sec
- ADC cutoff for severe fibrosis < 1.21 X 10-3 mm 2 / sec

8) MR Perfusion

- Progressive liver fibrosis gradually obliterates normal intrahepatic vessels and sinusoids and slows passage of blood through the parenchyma.
- Advanced fibrosis had increased absolute arterial blood flow, arterial fraction, distribution volume, and mean transit time.

9) MR Elastography

- New MR imaging technique that noninvasively quantifies the stiffness of the liver as well as other organs by analyzing the propagation of mechanical waves through tissue.
- In chronic liver disease, progressive deposition of interconnecting collagen fibers throughout the liver produces a lattice-like framework that increases parenchymal rigidity.

❖MRI VS US ELASTOGRAPHY

Advantages of MR Elastography:

1) Does not require an acoustic window and is suitable for evaluation of obese patients.
2) Operator independent.
3) The whole liver is visualized and the stiffness of the entire liver can be evaluated.

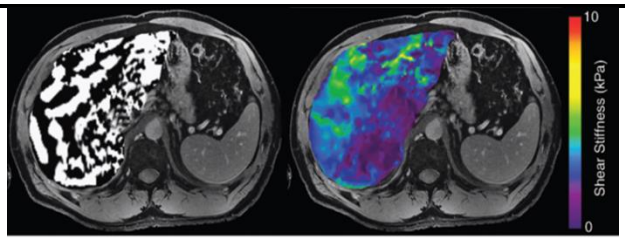
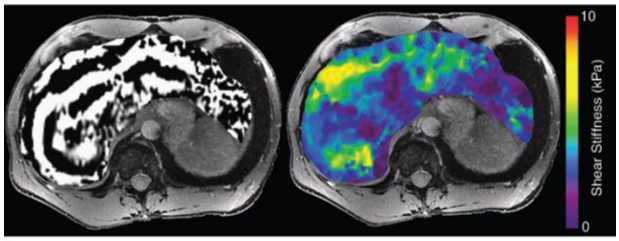
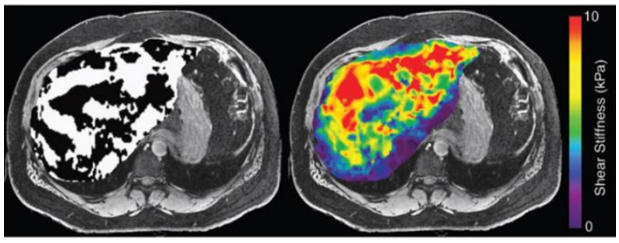
Limitations of US-based transient elastography

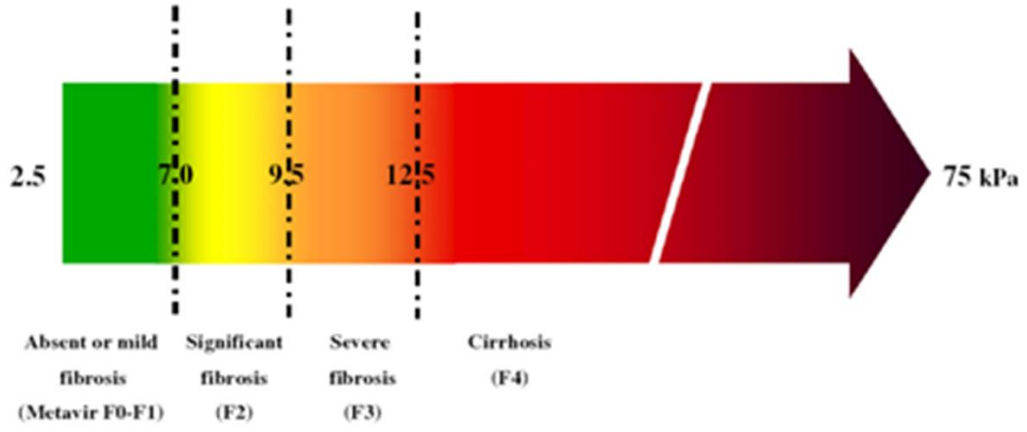
1) Requires an acoustic window and examinations frequently fail in obese patients and those with ascites.
2) Operator dependent
3) Assesses only a small volume of the liver near the surface, potentially introducing sampling variability

Limitations of MR elastography

1) MR elastography measures a surrogate of liver fibrosis (tissue stiffness) rather than fibrosis itself.
   - The relationship between fibrosis and stiffness is nonlinear, and predicting the histologic fibrosis stage from the stiffness value may be challenging, particularly in patients with early stages of fibrosis.
2) MR elastographic assessment of liver fibrosis may be confounded by a variety of factors expected to alter liver stiffness, including:
   - hepatic inflammation
   - Steatosis
   - Hepatic vascular congestion
   - Cholestasis
   - Portal hypertension
3) Other limitations of MR elastography are technical.

- Motion-sensitizing gradients are applied during the image acquisition.
- MR images are acquired with a gradient-echo sequence as the waves propagate through the liver.
- The velocity and wavelength of the waves propagating in the abdomen depend on the stiffness of the tissue (velocity and wavelength increase with greater tissue stiffness)
- Thus, images of the propagating waves can be used to estimate the stiffness.

No fibrosis	 a.
Mild fibrosis	 b.
Marked fibrosis	 c.



D- CIRRHOSIS

- Chronic liver disease characterized by:
    - Diffuse parenchymal necrosis + Fibrosis + Regenerative nodule

Classification based on morphology

- Micronodular (Laennec) cirrhosis
- Macronodular (postnecrotic) cirrhosis
- Mixed cirrhosis

Etiology

- Chronic viral hepatitis B/C
- Alcohol => Alcoholic steato-hepatitis (ASH)
- Non-alcoholic fatty liver diseases (NAFLD):
    - Steatosis => Nonalcoholic steato-hepatitis (NASH) => Cirrhosis
- Biliary:
    - Primary biliary cirrhosis
    - Primary sclerosing cholangitis
- Metabolic:
    - Hereditary haemochromatosis
    - Wilson disease
    - Alpha-1-antitrypsin deficiency
- Vascular disease (rare):
    - Congestive hepatomegaly (right heart failure)
    - Budd-Chiari syndrome, hepatic veno-occlusive disease

Imaging findings:

i. **Morphological changes (see before)**
ii. **Parenchymal changes (see before)**
iii. **2ry signs of portal hypertension (see before)**
iv. **2ry signs of hepatocellular failure (see before)**
v. **Stepwise carcinogenesis of HCC (see LIRADS):**
    a. **Regeneration nodule**
    b. **Dysplastic nodule**
    c. **HCC focus**
    d. **Small HCC**
    e. **Large HCC**
    f. **Advanced HCC**

E- Hepatic Schistosomiasis

- Schistosomiasis is the result of infection by blood fluke (trematode worm) of the Schistosoma species:
  - S. haematobium
  - S. mansoni
  - S. japononicum

Schistosomiasis can manifest as:

- Schistosomiasis dermatitis (swimmer's itch)
- Acute schistosomiasis (Katayama fever)
- Chronic schistosomiasis
  - Urinary schistosomiasis
  - Hepatosplenic schistosomiasis

Imaging findings:

i) Morphological changes:

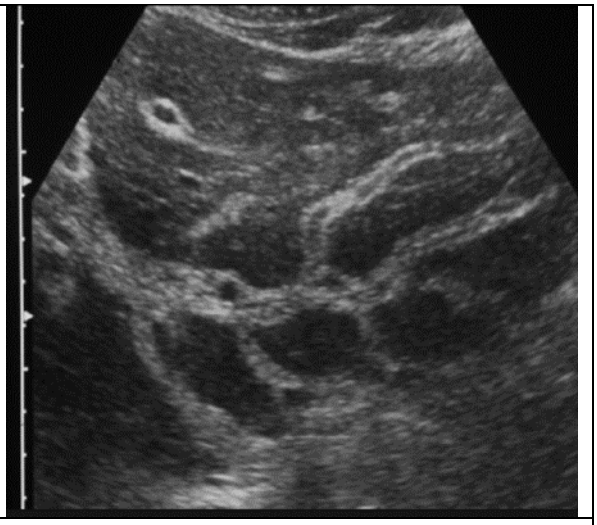
- Irregular hepatic contour
- Widening of fissures

ii) Parenchymal Changes:

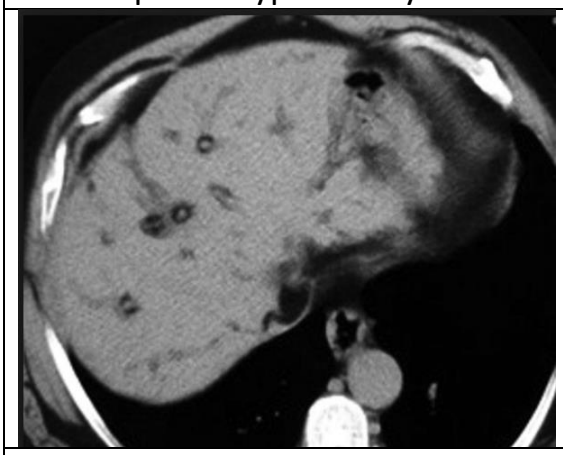
- Diffuse heterogeneous hepatic parenchyma
- Periportal fibrosis:
  - US: Hyperechoic thickened walls of portal venules = "clay-pipestem" pattern = Bull's-eye lesion
  - CT: Periportal hypodensity surrounding portal vein branches
  - T2: Periportal hyperintense signal
  - +C: Hypovascular with delayed contrast enhancement
- Calcification:
  - Calcified septa and fibrosis = turtle back sign
  - Capsular calcification

iii) 2ry sign of Portal hypertension (see before)

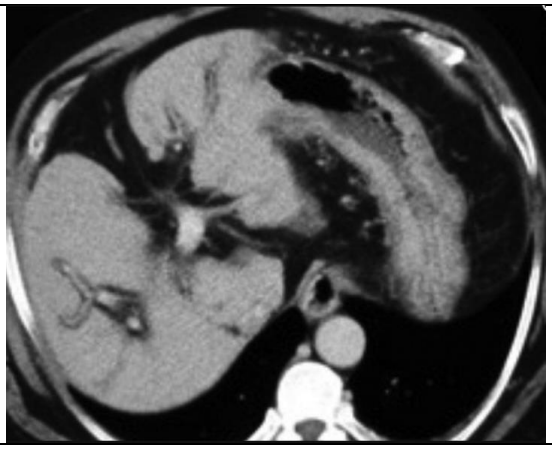
CT: Periportal hypodensity



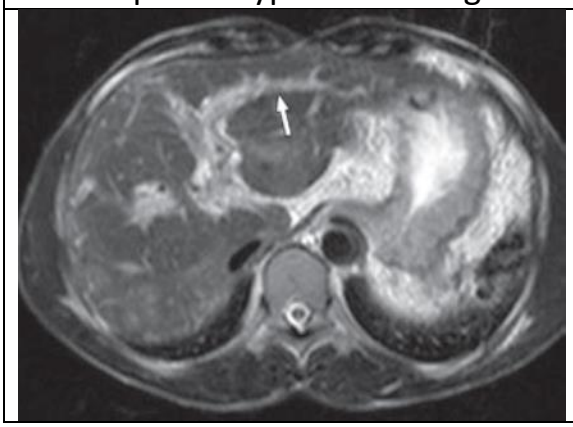
CT: Widened fissures



T2: Periportal hyperintense signal

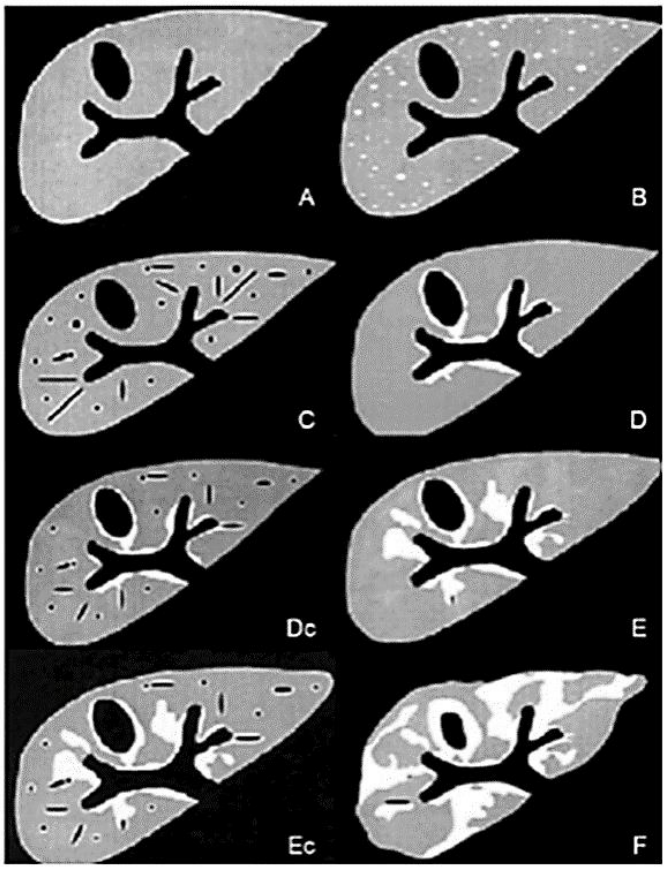


T1+C: Delayed contrast ehancement



❖ WHO patterns for US assessment of Schistosomiasis-related morbidity

A: normal liver structure
B: "starry sky" (diffuse echogenic foci)
C: highly echogenic "ring echoes", which correspond to the "pipe stems" seen in a scan perpendicular to the one where rings are seen
D: highly echogenic "ruff" around portal bifurcation and main stem
Dc: combined D and C patterns
E: highly echogenic "patches" extending from the main portal vein and branches into the parenchyma
Ec: combined E and C patterns
F: highly echogenic "bands" and "streaks", extending from the main portal vein and its bifurcation to the liver surface, causing retraction of the organ surface.



F- Hepatic Sarcoidosis

- Sarcoidosis is a common, chronic, non caseating granulomatous, multisystem disease of unknown origin.

- Age: Middle age
- Sex: F>M

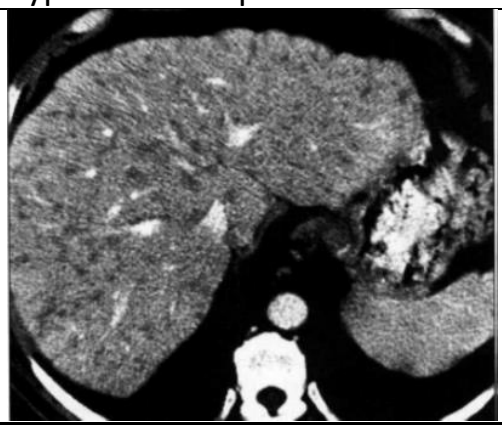
Best diagnostic clue:

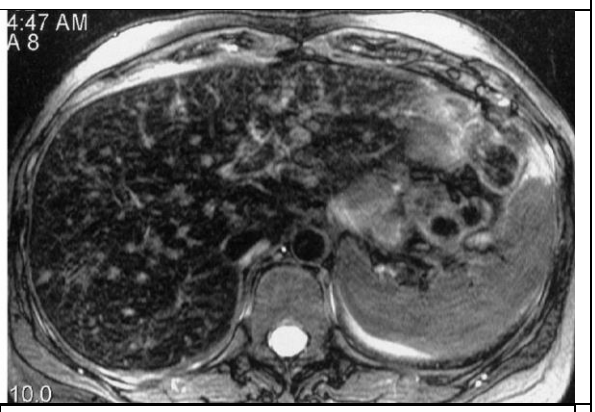
- Liver biopsy showing diffuse small noncaseating granulomas

Imaging findings:

- Hepatomegaly:
  - Diffuse heterogeneous parenchymal or multinodular pattern
- Splenomegaly:
  - Diffuse heterogeneous parenchymal or multinodular pattern
- Splenic nodules are larger and more common than hepatic nodules
- Multiple lymphadenopathy

Hepatomegaly + Multiple small hypodense heaptic focal lesions





Splenomegaly + Multiple small hypodense spelnic focal lesions

G- Hemochromatosis

Primary	Secondary
Hereditary autosomal recessive disease	Parenteral administration of iron (eg, repeated blood transfusions)
Increases intestinal absorption with normal dietary intake of iron	Dietary iron overload
Iron excess deposits in parenchymal cells, preferentially in the liver, pancreas, heart, pituitary gland, thyroid, and synovium	Iron is predominantly deposited in the reticuloendothelial system (Kupffer cells) (Liver + Spleen + BM)
Leads to cellular damage, organ dysfunction, and malignancy	Less toxic
If untreated, may progress to cirrhosis, hepatocellular carcinoma, diabetes, and cardiac dysfunction	Secondary increased absorption of normal dietary iron intake: liver diseases (alcoholic hepatitis and hepatitis C), myelodysplastic syndrome, anemias, other chronic and oncologic diseases

Hepatic biopsy

- Standard method for diagnosis
- Quantify iron overload
- Determine the prognosis on the basis of the level of hepatopathy
- Monitor the evolution of the disease and the effects of treatment.
- Susceptible to sampling error

❖Imaging Findings:

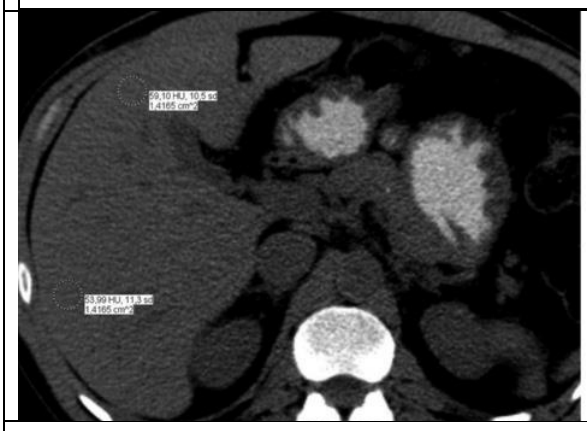
US:

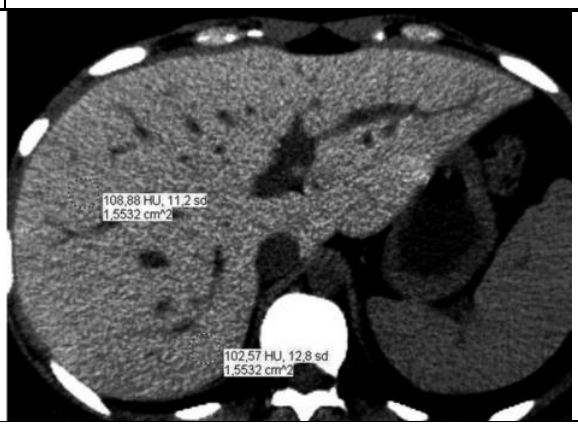
• No role in the diagnosis of hepatic iron overload

NECT

- Homogeneously increased liver density 75-135 HU (Normal 45-65 HU)
- DD: Wilson disease, colloidal gold treatment, and long-term administration of amiodarone

Normal Liver	Hemochromatosis

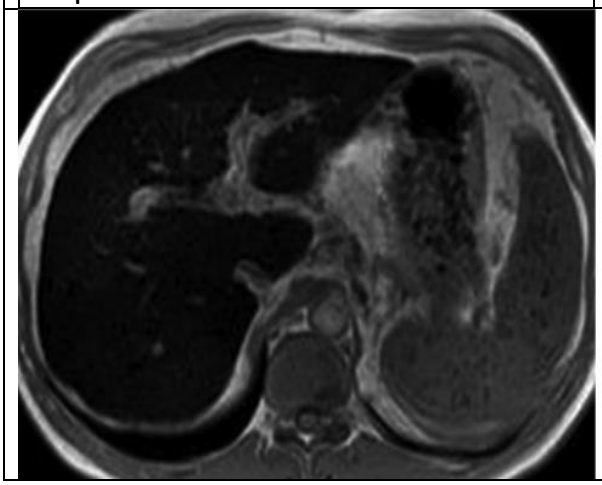


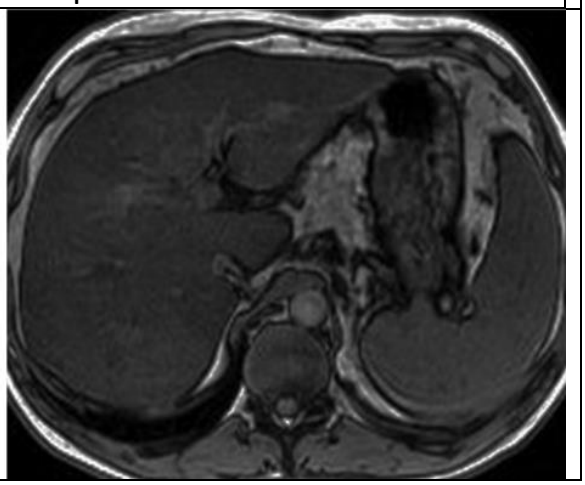


MRI:

- T1: Decreased signal in liver
- T2: Marked signal loss in liver
- In-phase: Decreased signal in the affected tissues compared with the out-phase image. (Opposite to steatosis)

In-phase	Out-phase



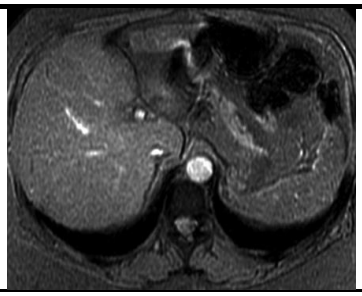
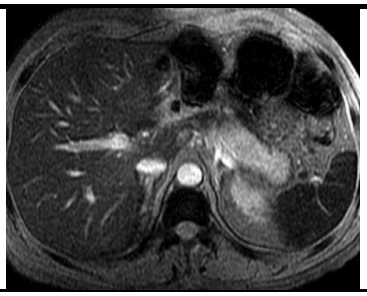
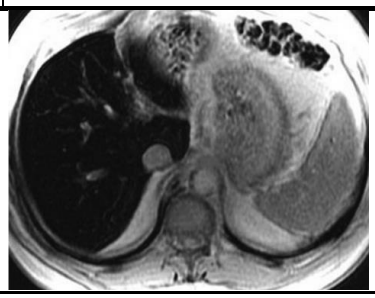


❖Iron Quantification with MRI

T2* GRE

- Signal intensity ratios of liver/muscle or liver/fat
- Establishes direct correlation with liver iron content

Normal:	Mild Hemochromatosis	Severe
Liver SI > Muscle SI	Liver SI < Muscle SI	Liver SI <<< Muscle SI

❖Iron Overload Patterns

Primary Hemochromatosis	Secondary Hemochromatosis
Parenchymal pattern of iron overload	**Reticuloendothelial pattern of iron overload**
Liver + Pancreas	Liver + Spleen + BM
Normal Spleen and BM	Normal pancreas

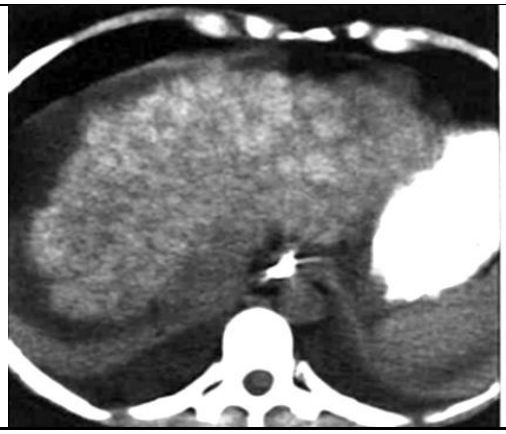
H- WILSON DISEASE

= Hepatolenticular Disease

- Autosomal recessive disorder in which copper (Cu) accumulates pathologically primarily within liver & subsequently in neurologic system & other tissues

Imaging findings:

- Hepatomegaly:
  - No characteristic features
  - Diffuse homogenous hyperdense
  - Diffuse homogenous hypodense (associated fatty infiltration)
  - Multinodular pattern
  - Cirrhosis with multiple hyperdense regeneration nodules

Diffuse homogenous hyperdense	**Cirrhosis with multiple hyperdense regeneration nodules**
	

I- Glycogen Storage Diseases

- Inherited autosomal-recessive disorders and are characterized by absence or deficiency of one of the enzymes responsible for producing or metabolizing glycogen.

- Different subtypes of glycogen storage disease involve the liver, musculature, hematopoietic system, myocardium, and kidneys.
- Types Ia (von Gierke), Ib, II (Pompe), III (Forbes), IV (Anderson), VI, and IX have the cardinal symptom of hepatomegaly.

Clinical picture:

- Children + Hepatomegaly + Hypoglycemia + Growth retardation + Disproportional distribution of body fat

J- Lipid Storage Diseases

Diverse family of diseases related by their enzymatic deficiency of a lysosomal hydrolase:

- GM1 gangliosidosis (β-galactosidase)
- GM2 gangliosidosis (β hexosaminidase)
- Gaucher disease (glucocerebrosidase),
- Niemann- Pick disease (sphingomyelinase),
- Fabry disease (α-galactosidase A),
- Schindler disease (α-galactosidase B)
- Metachromatic leukodystrophy (arylsulphatase A),
- Krabbe disease (galactosylceramide- β-galactosidase),
- Farber disease (ceramidase),
- Wolman disease (cholesteryl-esterhydrolase).

Imaging findings:

- Hepatomegaly:
  - Non-specific imaging finding
  - Diffuse low density due to lipid and cholesterol deposition
  - Leading to Fibrosis and cirrhosis

II- Hepatic Cystic Lesions

❖ **Developmental:** ○ **Hepatic cyst** ○ **Biliary hamartoma** ○ **Polycystic liver disease** ○ **Choledochal cyst** ○ **Caroli disease**
❖ **Inflammatory:** ○ **Pyogenic abscess** ○ **Amoebic abscess** ○ **Candidiasis abscess** ○ **Hydatid cyst**
❖ **Trauma: (see trauma)** ○ **Hematoma** ○ **Biloma** ○ **Pseudo-aneurysm**
❖ **Neoplasms:** ○ **Biliary cystadenoma** ○ **Biliary cystadenocarcinoma** ○ **Mesenchymal Hamartoma** ○ **Embryonal sarcoma** ○ **Cystic metastasis** ○ **Giant hemangioma** ○ **Necrotic HCC**

A- Developmental

1) Simple (Bile duct) Cyst

- Simple hepatic or congenital cyst are benign developmental lesions
- Not communicate with the biliary tree.
- 1%–3% of routine liver examinations.

- Solitary
- Multiple :
  - Polycystic kidney (ADPCKD)
  - Von-Hippel Lendu (VHL)

US:

- Well defined round or ovoid lesion. No evident wall.
- Anechoic
- Posterior enhancement
- Subtle lateral acoustic shadows.
- Color Doppler avascular

CT:

- Well defined round or ovoid lesion. No evident wall.
- NCCT: Homogeneous and hypoattenuating content <20HU
- CECT: No enhancement of the wall or its content.

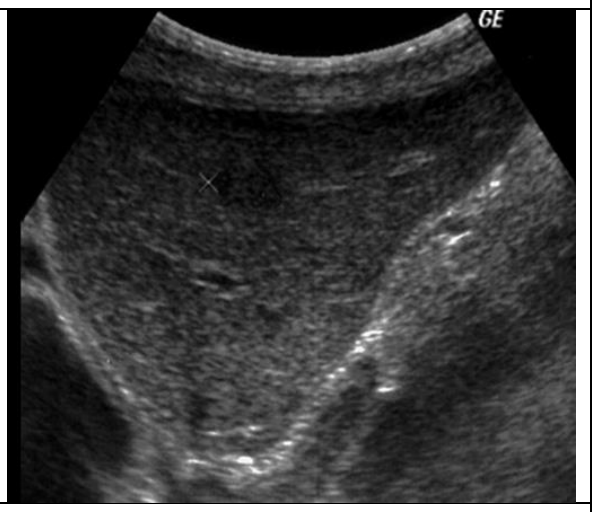
MRI:

- Well defined round or ovoid lesion. No evident wall.
- T1 WI: homogenous very low SI
- T2 WI: very high SI
- Heavily T2: the signal increases further, nearly reaching the signal intensity of fluid
- Gd: No enhancement

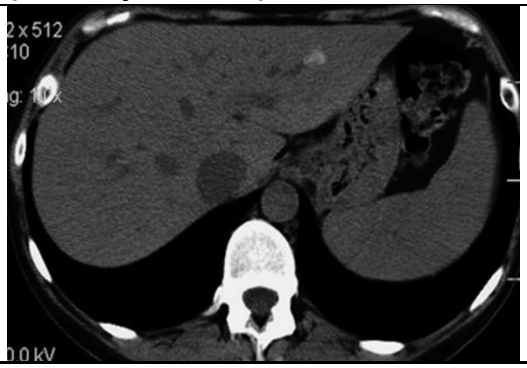
2) Complicated Cysts

- Higher attenuation values are present in cyst with hemorrhage or inflammation inside

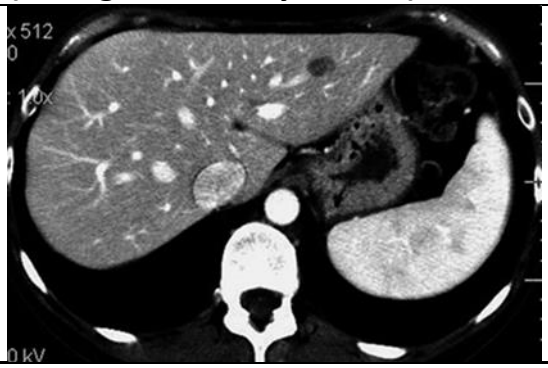
US: Heterogeneous hyperechoic lesion



NCCT: hyperdense lesion (Density > 50HU)



CECT portal phase: No enhancement (Change in density <10HU)



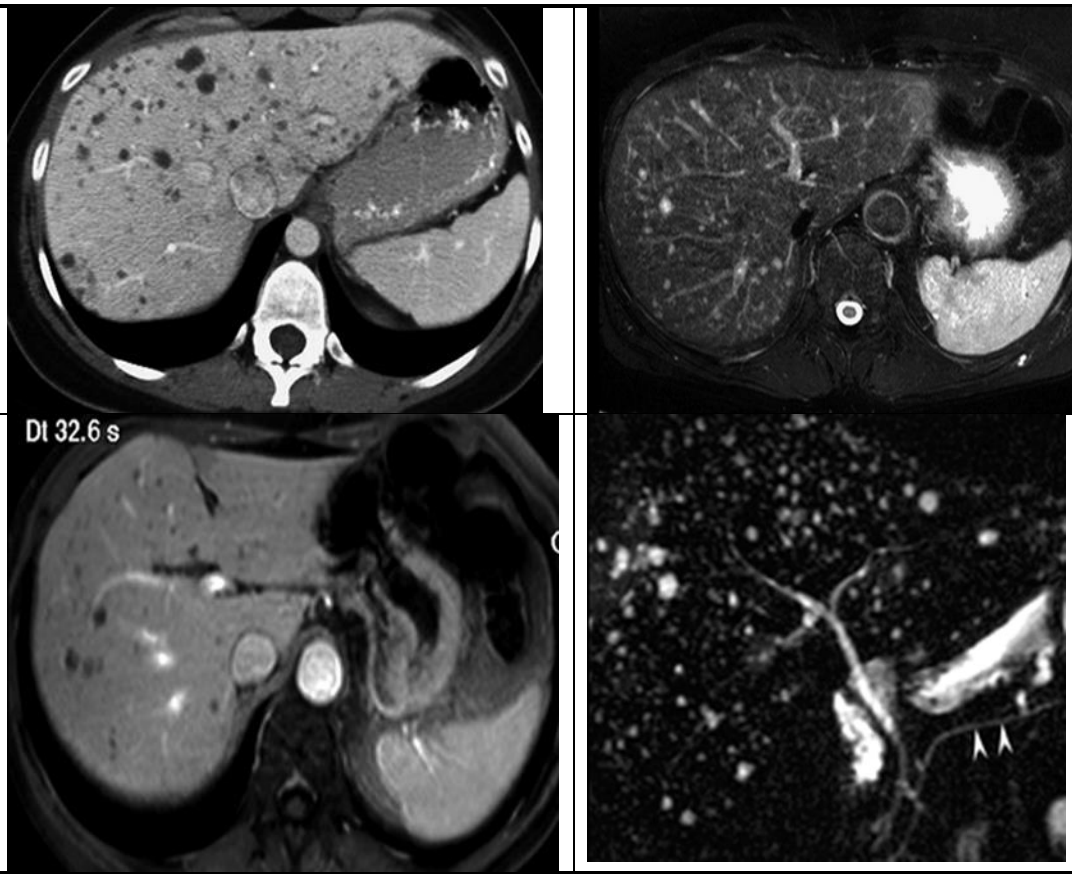
3- Biliary Hamartoma

- Also known as biliary microhamartomas or von Meyenburg complex
- Arise owing to failure of involution of embryonic bile ducts.
- 0.69%–2.8%

Imaging findings:

- Multiple round focal lesions of nearly uniform size
- Scattered throughout the liver.
- Typically measuring < 1.5 cm in diameter
- No renal involvement
- No communication with the billiary tree.

- NCCT: Hypodense
- T1: hypointense compared with liver parenchyma
- T2: markedly hyperintense
- Heavily T2: the signal increases further, nearly reaching signal of fluid
- +C: No enhancement

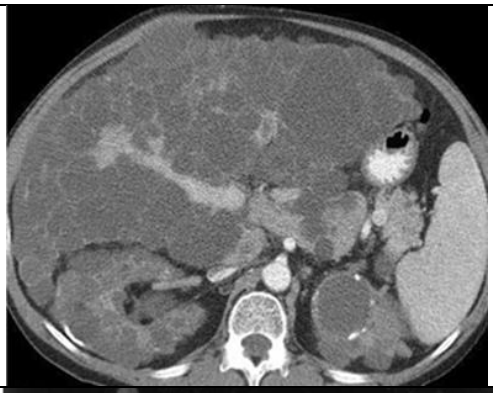
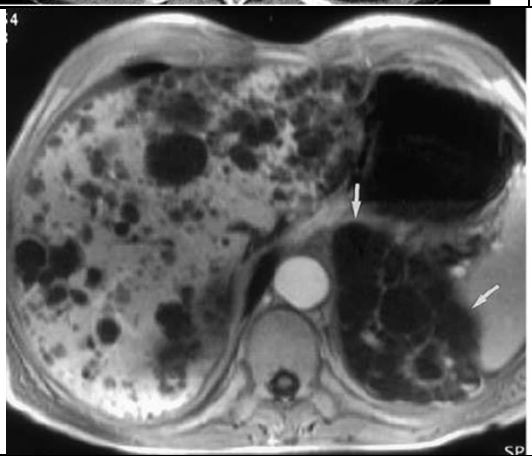
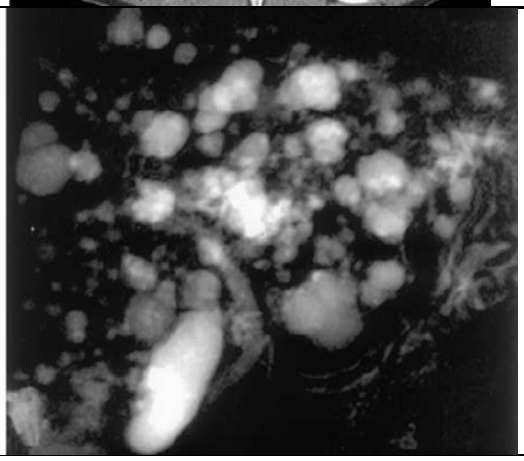


4- Polycystic Liver Disease

- Autosomal dominant (AD) disorder
- Associated with autosomal dominant polycystic kidney disease (ADPKD).

- Hepatomegaly
- Diffusely cystic liver
- Cysts varying from less than 1 mm to 12 cm or more in diameter
- Calcification of the walls of hepatic cysts and renal cysts as a result of prior hemorrhage and inflammation is occasionally detected.
- No communication with the biliary tree.
- ± association with renal polycystic disease

- NCCT: hypodense
- T1: Low signal
- T2: High signal
- Heavily T2: very high signal like fluid
- +C: No enhancement

NCCT	CECT
	
	

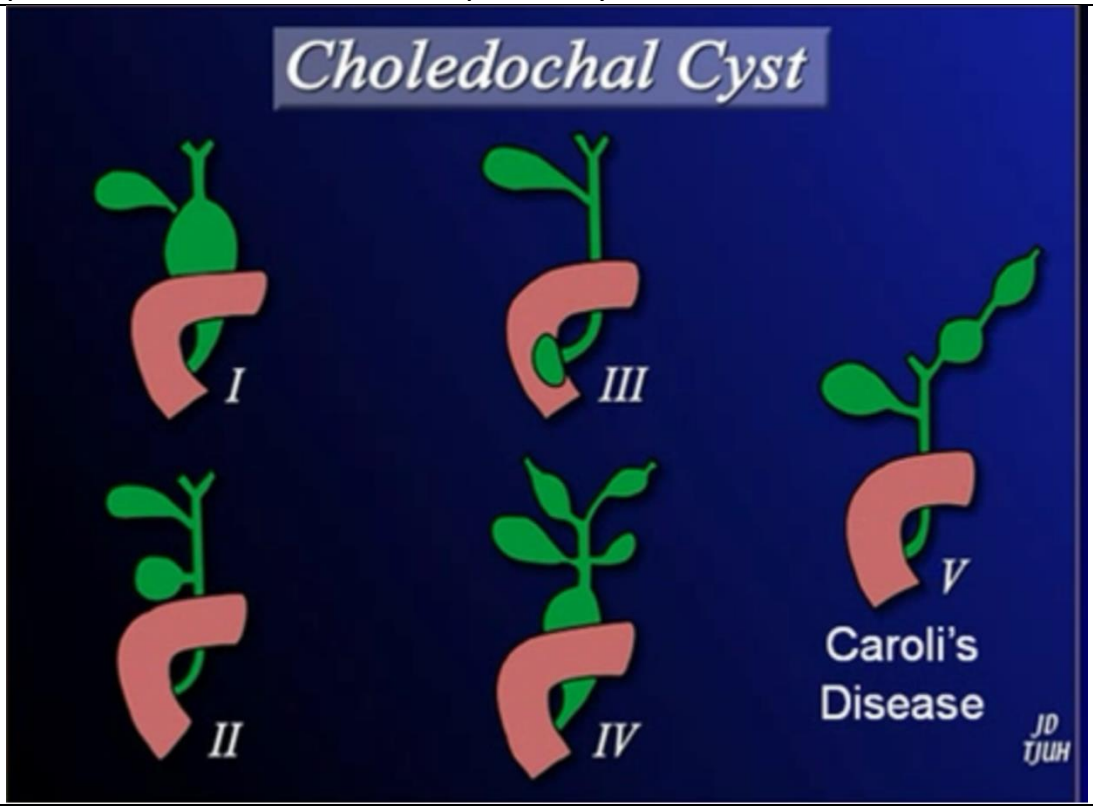
5- Choledochal Cyst

- A choledochal cyst is a rare congenital dilation of the hepatic duct of the liver and is due to a ductal plate malformation of the large bile ducts.

❖ Todani classification of choledochal cysts:

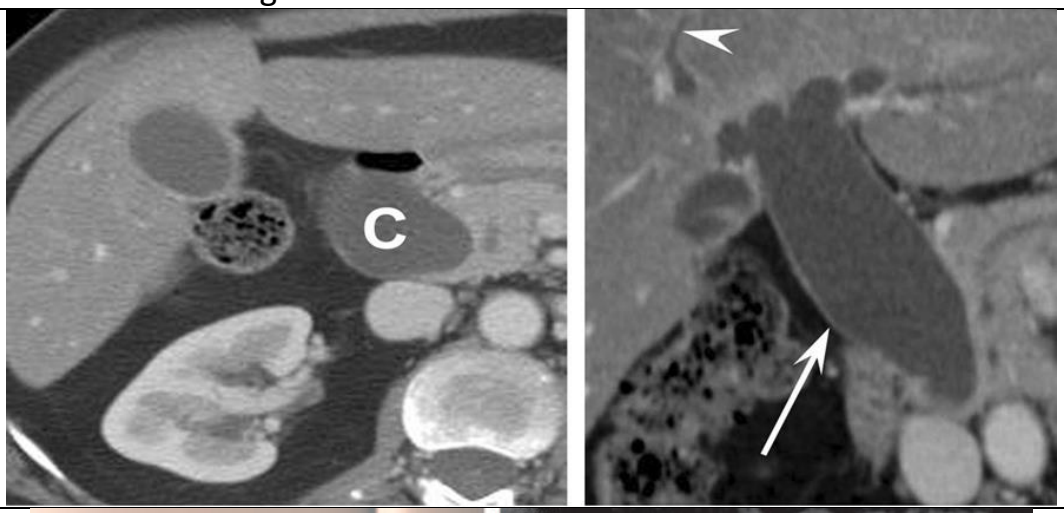
Type 1 : Extrahepatic
- 1a : Cystic dilatation of CBD
- 1b : Segmental dilatation of CBD
- 1c : Fusiform dilatation of CBD

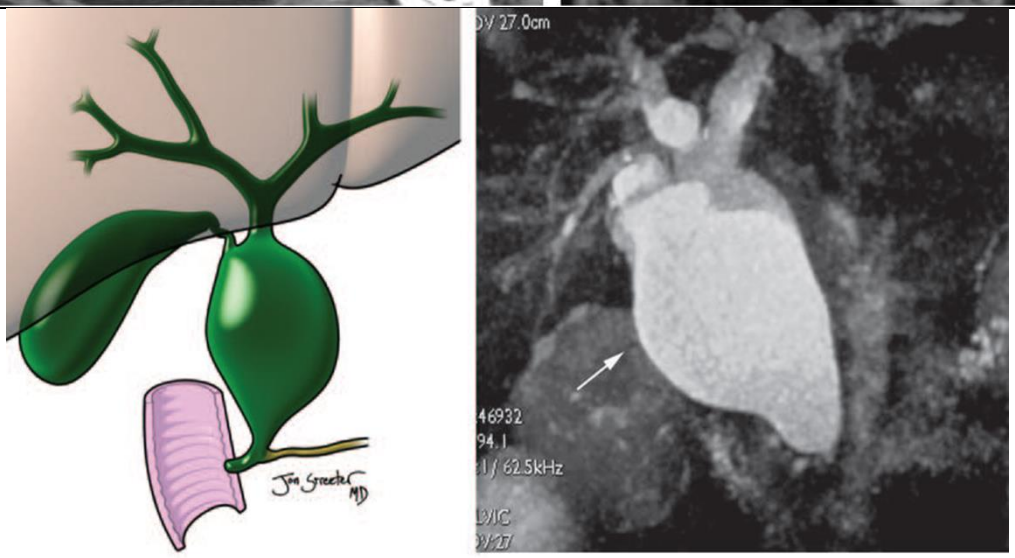
Type 2 : True diverticulum of CBD
Type 3 : Dilatation of distal intramural segment of CBD (Choledochocele)
Type 4a : Multiple dilated Extra & Intra hepatic
Type 4b : ,, ,, Extra hepatic only
Type 5 : ,, ,, Intra hepatic only



❖ Type I: Choledochal cyst

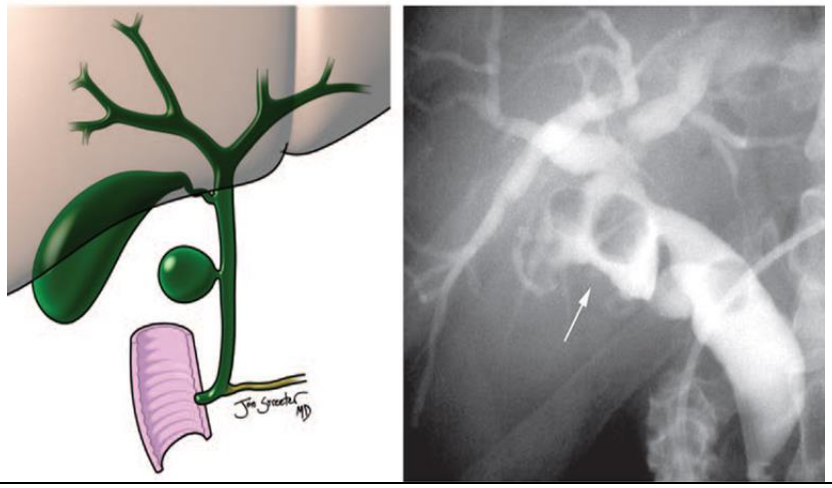
- Fusiform cystic dilatation of the CBD
- Cystic lesion at porta hepatic.
- Fluid echogenicity/density/intensity in US, CT, or MR.
- Communication with biliary tree.
- Not communicating with GB.
- Intraluminal sludge or stone.





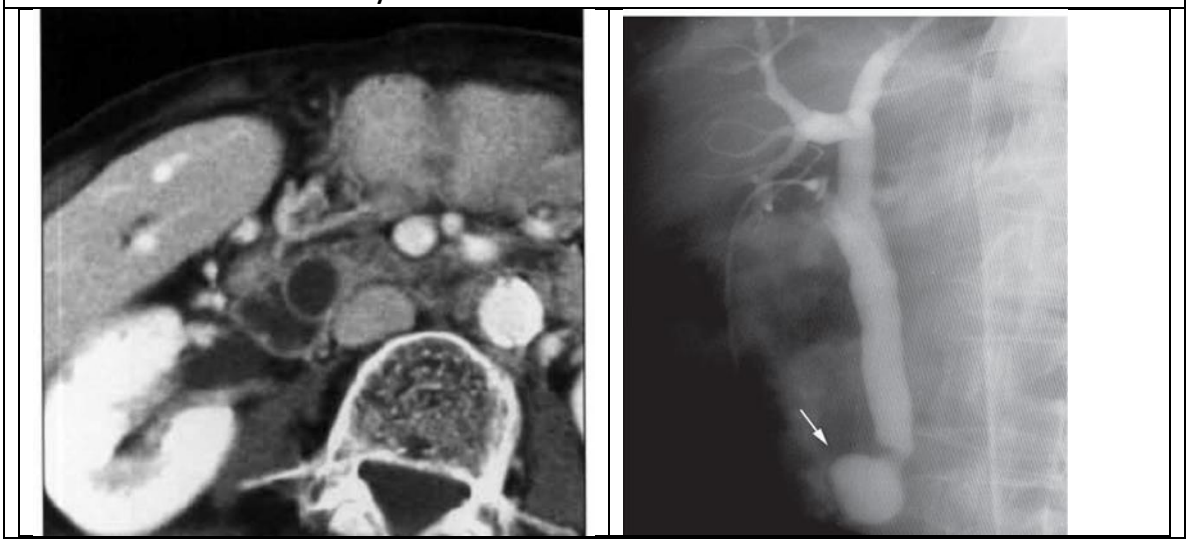
❖ Type II: Choledochal cyst

- Well defined outpouching diverticulum of the CBD
- Cystic lesion at porta hepatic.
- Water echogenicity/density/intensity in US, CT, or MR.
- Communication with billiary tree.
- Not communicating with GB.
- Intraluminal sludge or stone



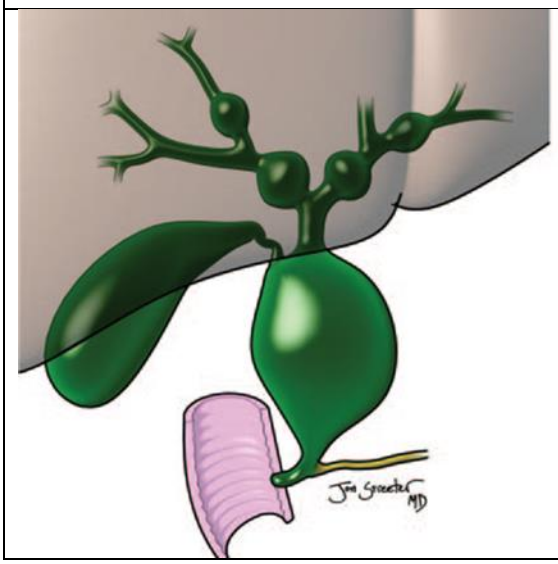
❖ Type III: Choledochocele

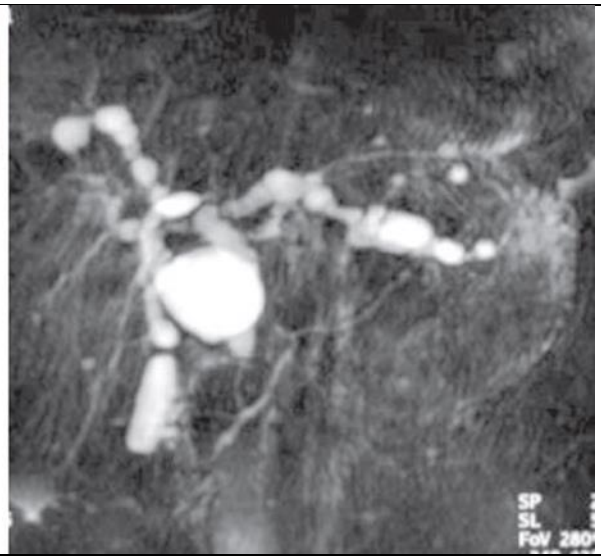
- Cystic lesion within the medial wall of the duodenum
- The rest of the biliary tree was normal.



❖ Type IV: Choledochal Cyst

- Intra and extra-heaptic biliary dilatation
- No obstruction



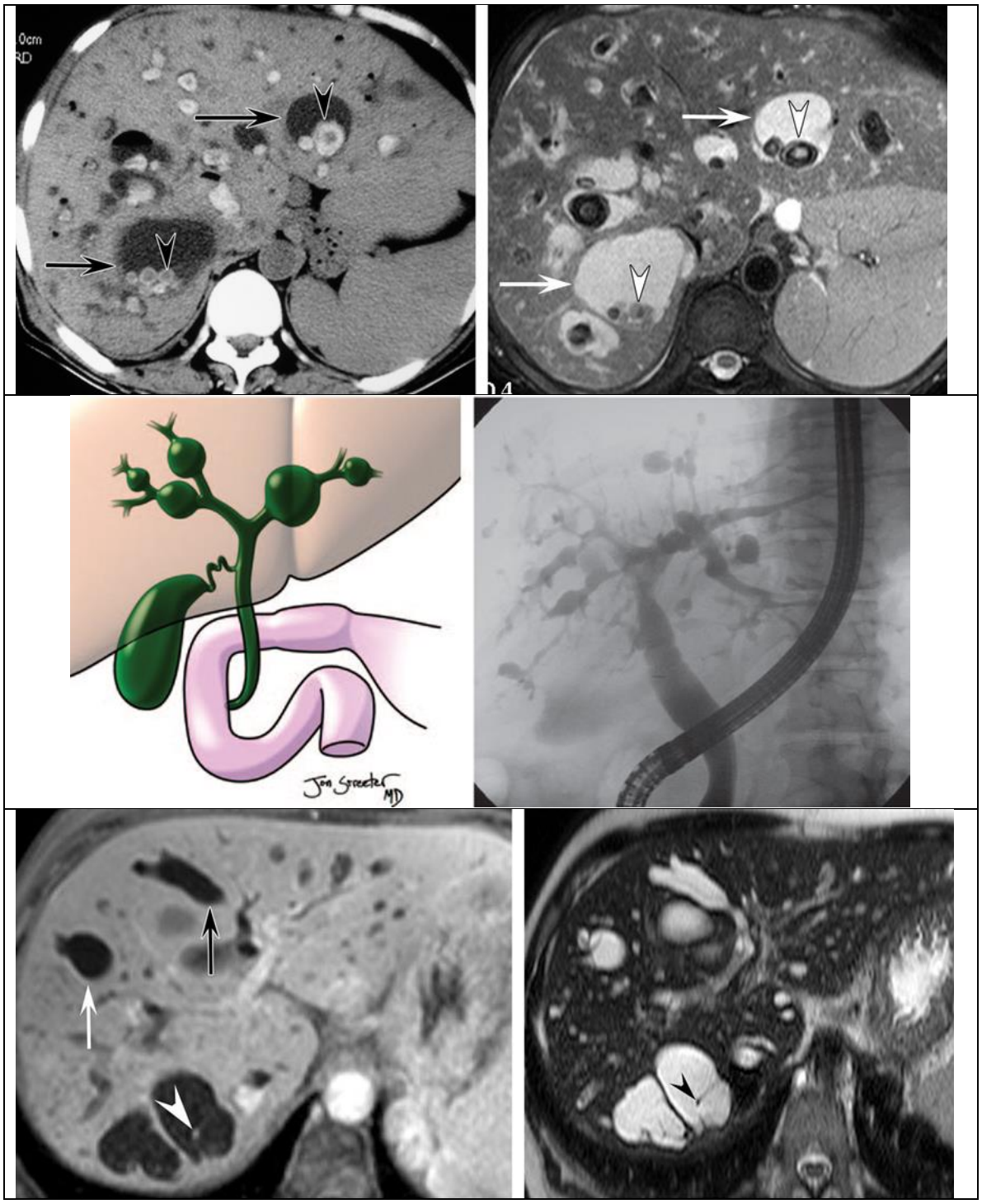


❖ Type V: Caroli's disease

- Cystic (Saccular) intra-hepatic biliary dilatation.
- Multiple branching, tubular structures.
- Communicating with billiary tree.
- Extending to periphery of liver.
- Radiate towards pota hepatis.
- Central dot sign: Portal radicles are partially or completely surrounded by abnormally dilated & ecstatic bile ducts.

Caroli Syndrome

- Caroli disease + Congenital hepatic fibrosis


0cm
RD
Jon Greeter MD

B- Inflammatory Cystic Lesions

1- Pyogenic Abscess

Aetiology: 5 Major routes:

1) Biliary route: due to ascending cholangitis - biliary obstruction.
2) Portal vein route : due to phlebitis from :
    - Appendicitis – Diverticulitis – IBD – Proctitis.
3) Hepatic artery route: Septicemia- bacterial endocarditis.
4) Direct extension : from adjacent organs :
    - Pneumonia – Pyelonephritis - Subphrenic abscess.
5) Traumatic route: Blunt or penetrating.

Causative Organism:

- Most commonly caused by Clostridium species and gram-negative bacteria, such as Escherichia coli and Klebsiella, Enterococcus, and Streptococcus

Distribution:

- The abscesses related to the portal mechanism are usually single.
- The abscesses related to the biliary mechanism are typically multiple and scattered in both lobes

2 patterns:

- Pyogenic microabscesses (2 cm)
- Large macroabscess

Imaging Findings:

- Well defined cystic lesion
- Thick wall marginal enhancement
- Perilesional hypodense edema rim
- Double target sign:
    - Hypodense central pus area
    - surrounded by an inner enhancing ring of granulation tissue and outer hypodense zone of inflammatory edema
- Cluster sign: smaller lesions surrounding a large abscess.
- Air bubble or Air fluid level.

Thick walled cystic lesion **Marginal enhancement** **Air bubble**	**Cluster sign: smaller lesions surrounding a large abscess.**
Multiple micro abscesses scattered in both hepatic lobes	**Double target sign = an inner enhancing ring of granulation tissue and outer hypodense zone of inflammatory edema**

2- Amoebic Abscess

Aetiology :

- Entamoeba histolytica is common parasite in Asia – Africa - South & Central America.

Distribution :

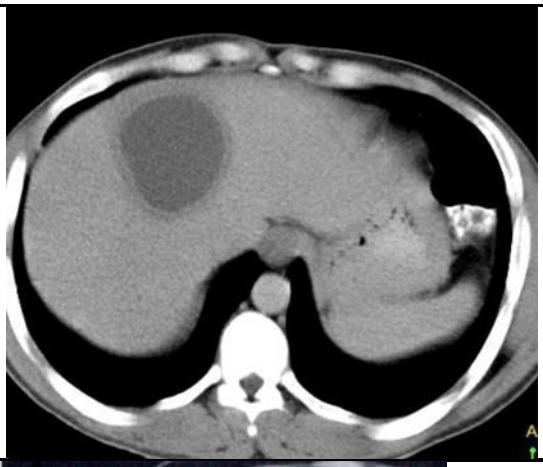
- Solitary
- Rt lobe > Lt lobe

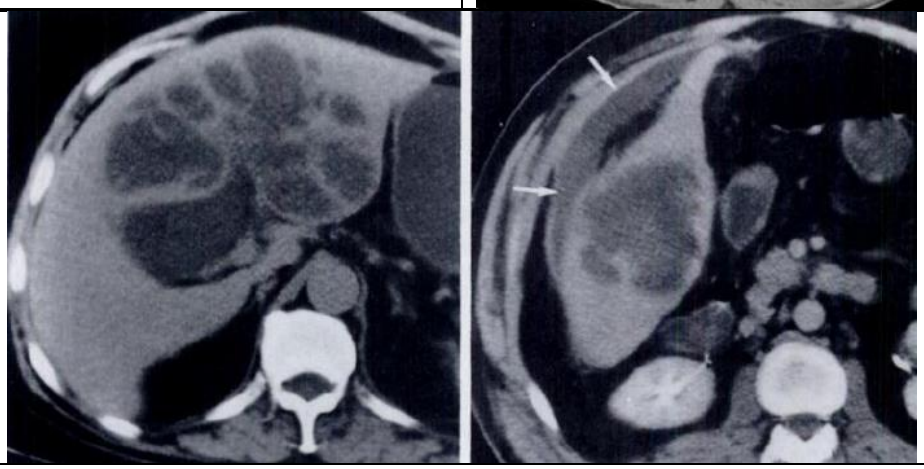
Imaging findings:

- A well-defined rounded lesion
- The central content CT attenuation = complex fluid (e.g. 10–20 HU).
- The central abscess cavity can show septations and/or fluid-debris levels
- An enhancing thick wall
- A peripheral zone of oedema

Extrahepatic abnormalities:

- Pleural effusion
- Perihepatic fluid collection
- Gastric or colonic involvement
- Retroperitoneal extension





3- Candidiasis Abscess

Aetiology :

- Candida in immunocompromized patients eg.: AIDS-organ transplantation- chemotherapy.

Distribution :

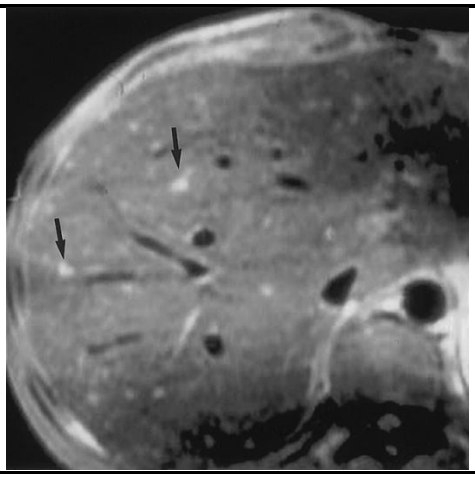
- Multiple: scattered in both heaptic lobes .
- Small : < 1cm

US: 3Major pattern:

1) Wheel within a wheel:
    - Central hypoechoic nidus: related to focal necrosis.
    - Inner echogenic ring: caused by inflammatory reaction.
    - Outer hypoechoic ring: caused by oedema.
2) Bull's eye:
    - Hyperechoic center surrounded by hypoechoic rim.
3) Uniform hypoechoic.

Imaging findings:

- Multiple, small, rounded cystic focal lesions.
- CT: hypodense
- T1: Low signal
- T2: high signal
- Calcification can be seen within the lesion.
- No enhancement



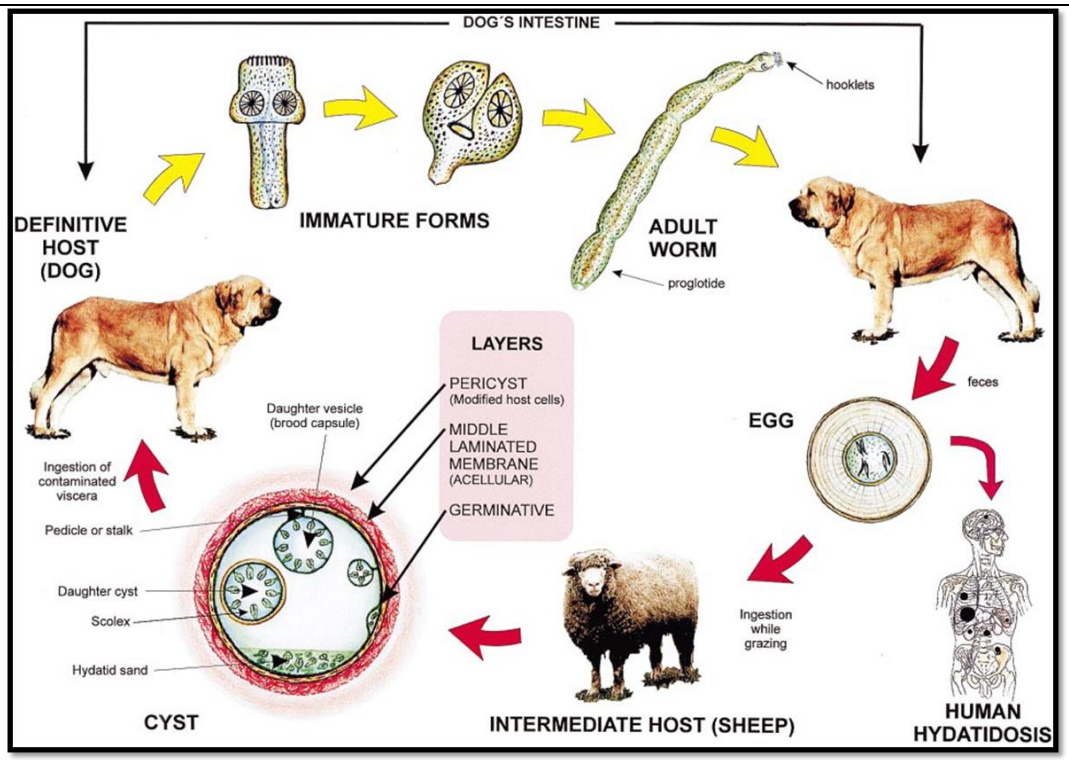
4- Hydatid Cyst

Aetiology :

1) Echinococcus granulosus : more common
2) ,, alveolaris : less common.

Life cycle :

- Definitive host : Dog
- Intermediate host : Human



Pathology :

- Cyst wall consists of 3 walls :
  Pericyst – Exocyst – Endocyst.

Complications :

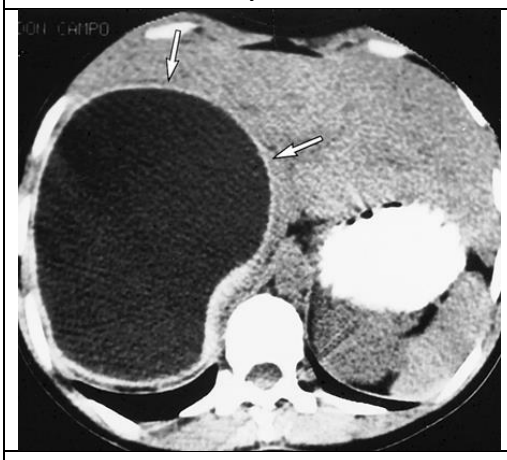
- Cyst rupture:
  - Peritoneal / Pleural / Pericardial cavity.
  - Biliary system => Obstructive jaundice.
  - Circulation => Anaphylaxis.
- Superinfection.

Imaging findings:

- Well defined cystic lesion.
- Multilocular > Unilocular.
- Daughter cysts: multiseptate cysts, arranged towards periphery
- Satellite cysts: located outside main lesion.
- Water lilly sign: Free floating membrane.
- Double rim sign: Pericyst and Endocyst.
- Calcification : Curvilinear ring – grossly diffuse

Type I: Simple Cyst

- Unilocular cyst

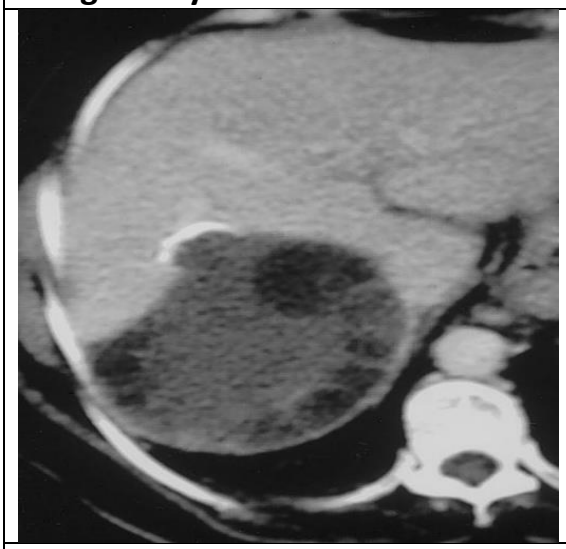


Type III: Calcified Cyst

Dead cysts with total calcification.

Type II: Cyst with Daughter Cyst(s) and Matrix

Daughter cysts



Free floating membrane

Type IV: Complicated Hydatid Cyst

C- Neoplastic Cystic Lesions

1- Biliary Cystadenoma

- Uncommon benign cystic tumor of bile ducts
- Age: Middle aged women
- Sex: F:m=4:1
- It is regarded as premalignant tumor.
- Malignant transformation 15% of cases.

Location:

- 55% occur in the right lobe
- 29% occur in the left lobe
- 16% occur in both lobes

Imaging findings:

- Large intrahepatic solitary complex cystic lesion
- Well defined with lobulated margins
- Multiloculated cystic lesion with internal septations.
- Calcification may be seen within the wall and the septa
- ± Hemorrhagic fluid ± Fluid / fluid level
- ± Low level echoes may be seen within cyst.

+C:

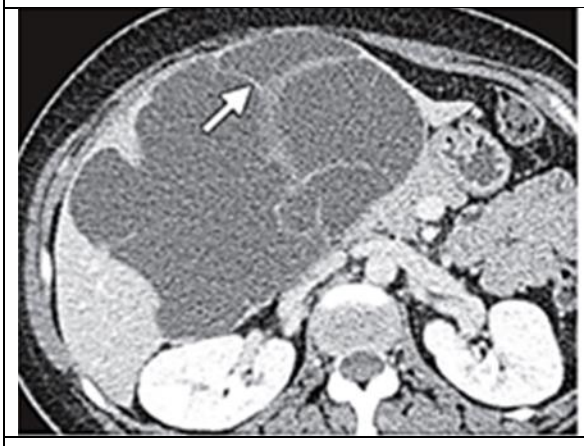
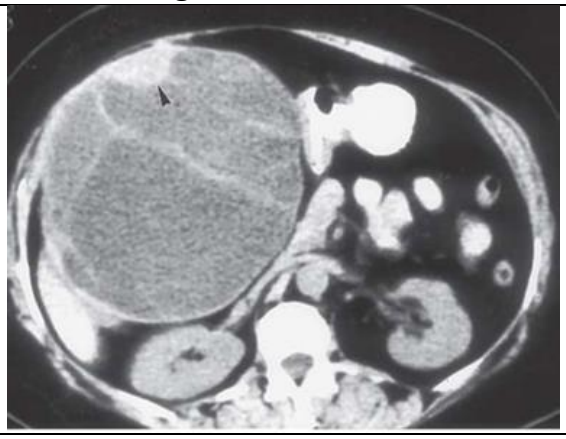
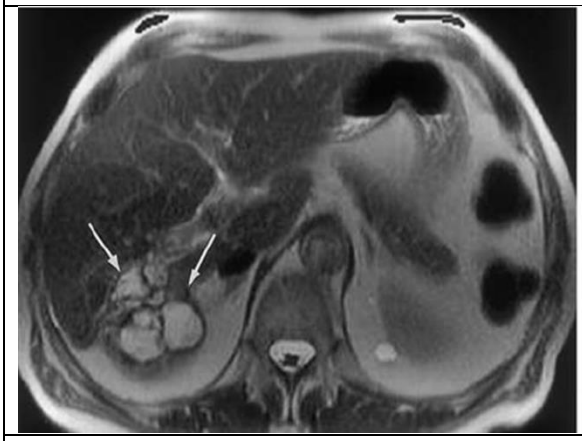
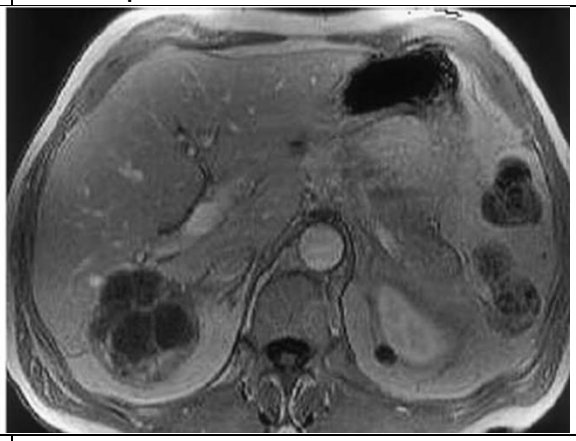
- The cystic part of lesion→ don't enhance
- Wall +Internal septation →Enhance

2- Biliary Cystadenocarcinoma

Malignant transformation of biliary cystadenoma :-

- **Thick septations.**
- **Mural nodule.**
- **Soft tissue mass.**
- **Amorphous calcification**.

- Differentiation between cystadenocarcinoma and cystadenoma is often difficult when based solely on imaging findings because of the overlap in radiologic features

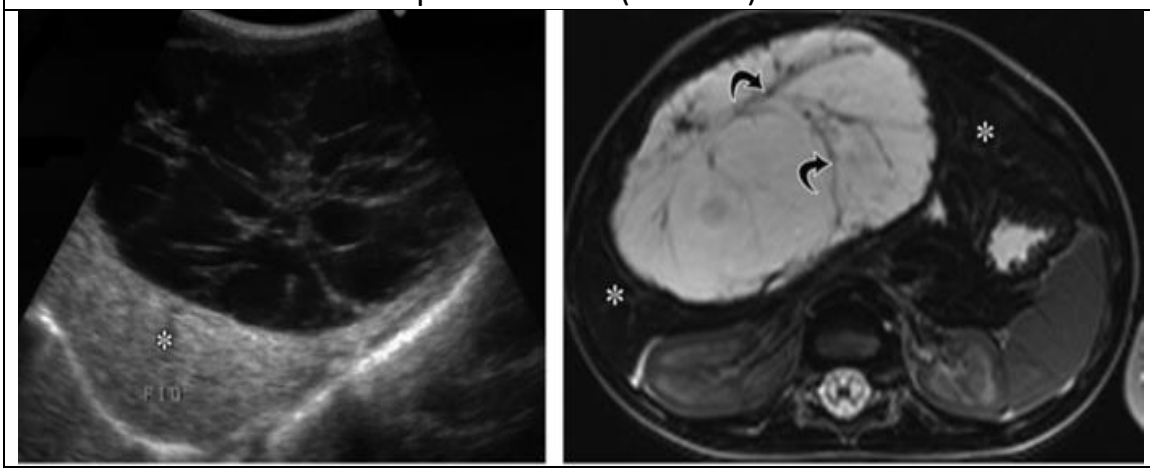
Multilocular cystic lesion + Thick enhancing internal septations	Multilocular cystic lesion + Thick enhancing internal septations + Enahcning mural nodule
	
T2: Multilocular cystic lesion	T1+C: Enhancing internal septations and capsule
	

3- Mesenchymal Hamartoma

- 2nd most common benign liver mass in children after infantile hemangioendothelioma
- Neonates < 2y

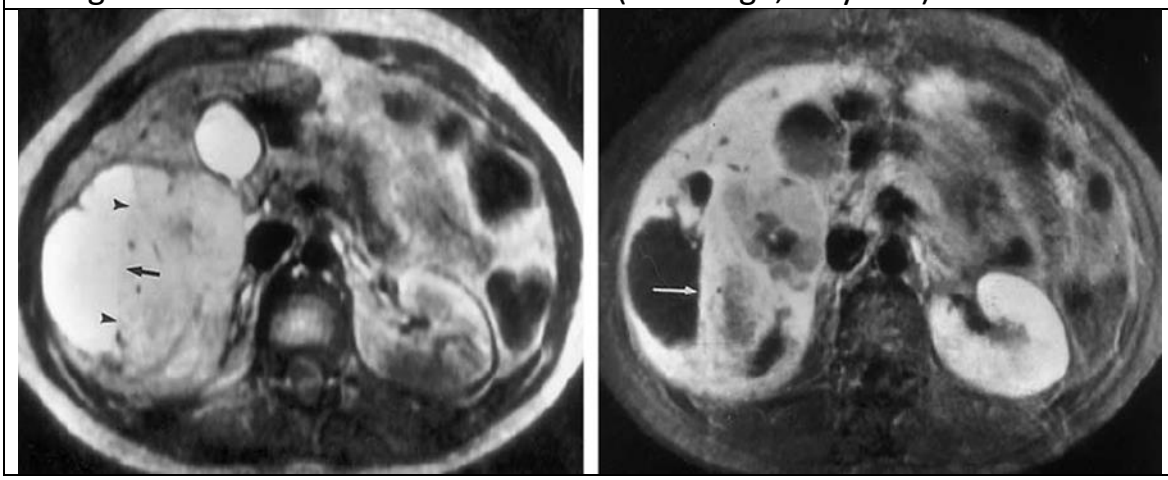
Imaging findings:

- Complex cystic mass + Septations
- The cystic components show fluid attenuation, low T1 and High T2.
- The stromal components are hypoattenuating to surrounding liver.
- Enhancement of the septa and solid (stromal) elements is observed



4- Undifferentiated Embryonal Sarcoma

- Rare malignant hepatic tumor
- Age: older children and adolescents (mean age, 12 years)



5- Cystic Metastasis

a) 1ry neoplasm has a cystic component:

eg.: Mucinous cystadenocarcinoma of colon – stomach – pancreas - ovary.

b) Metastasis undergoing central necrosis:

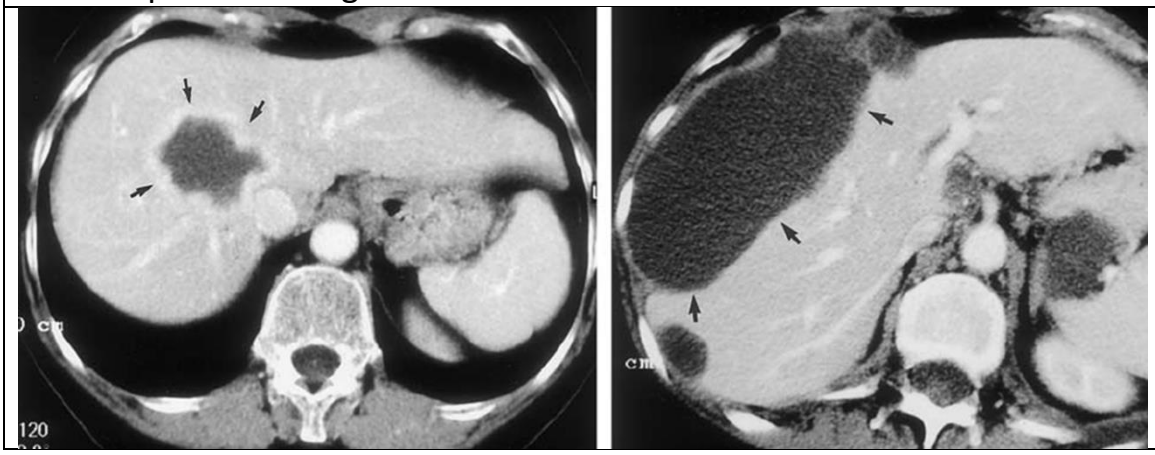
eg.: neuroendocrine tumors, sarcoma - certain subtypes of lung and breast - SCCa – leiomyosarcomas –melanoma -testicular carcinoma.

c) Systemic or locoregional TTT:

Treated liver metastases may also appear as multilocular cystic lesions with decreased attenuation at CT and increased signal intensity on T2. e.g: GIST after treatment with imatinib mesylate

Imaging findings:

- Multiple cystic lesions
- The attenuation of the cystic component is usually higher than that of pure fluid because of the presence of necrosis, mucin, or hemorrhage.
- Thick enhancing septations.
- Multiple enhancing mural nodules



III- Hepatic Solid Lesions

A- Adults:

- **Hemangioma**
- **Hepatic adenoma**
- **Fibronodular hyperplasia**
- **Hepatocellular Carcinoma**
- **Fibrolamellar Hepatocellular Carcinoma**
- **Cholangiocarcinoma (Discussed with biliary)**
- **Metastasis**

B- Pediatric:

- **Hepatoblastoma**
- **Hemangioendothelioma**
- **Mesenchymal hamartoma**
- **Embryonal sarcoma**
- **Metastatic disease (Neuroblastoma)**

A- Adult Solid Hepatic Focal Lesions

1- Hemangioma

- **Hemangioma is most common solid benign hepatic tumor.**

Hemangioma is associated with clinical syndromes including

- Klippel-Trenaunay-Weber syndrome
- Osler-Rendu-Weber disease
- Von Hippel–Lindau disease

US:

- Focal, homogeneous, hypovascular, echogenic lesions.
- However, this appearance is variable depending on the degree of fatty infiltration or fibrosis of the liver, which may result in a relatively hypoechoic appearance of the lesion.

MRI:

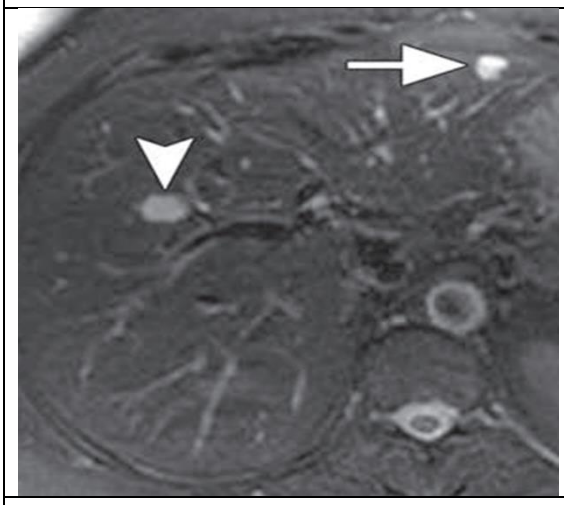
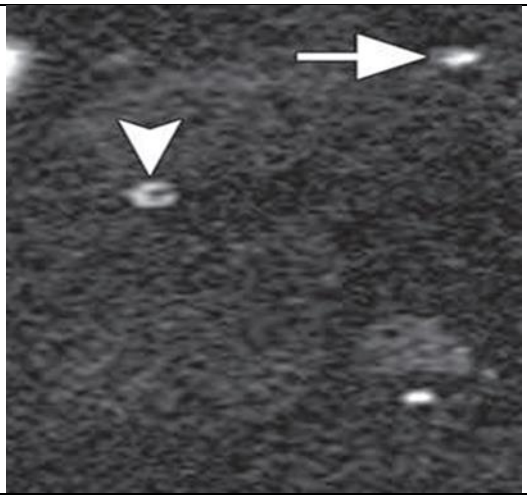
- T1: Low SI
- T2: marked high SI (light bulb)
- DWI: High signal due to T2 shine through effect
- ADCs of hemangiomas are
  - > Solid malignant lesions
  - < Cysts

3 Enhancement Patterns

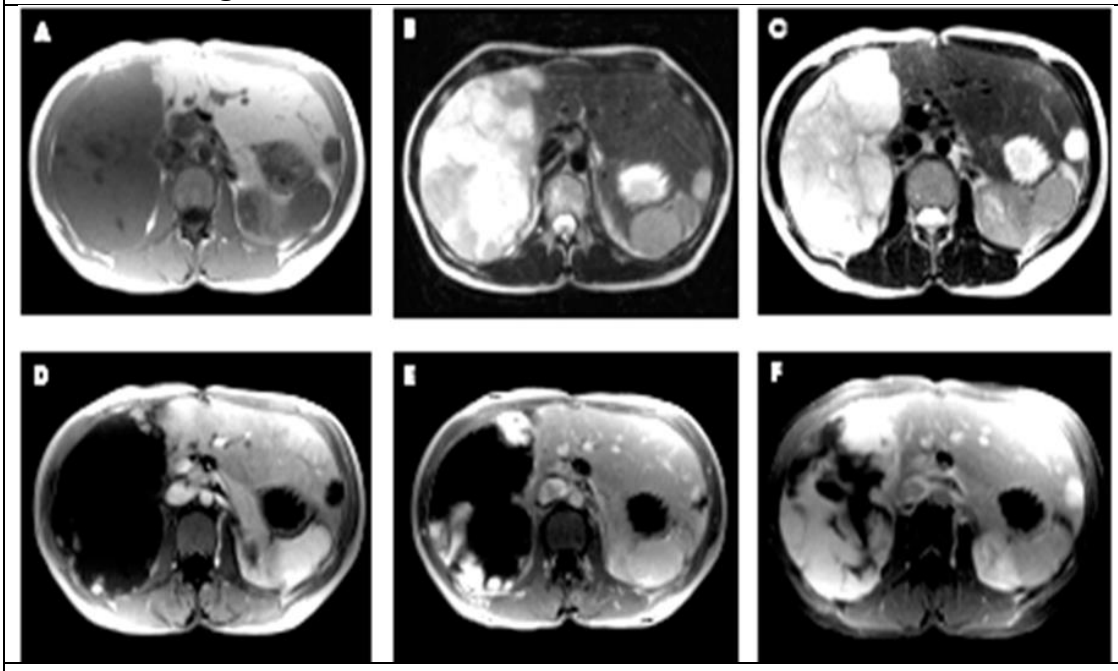
- **Pattern 1: (small capillary hemangiomas <1.5 cm)**
  - Immediate uniform enhancement
- **Pattern 2: most common pattern (77% of cases)**
  - A well-circumscribed hepatic mass with peripheral, nodular, and interrupted enhancement and progresses centripetally to uniform enhancement.
- **Pattern 3: (Giant hemangiomas >5 cm).**
  - Peripheral nodular enhancement with centripetal progression but persistent central hypointensity

Hepatobiliary specific contrast agent

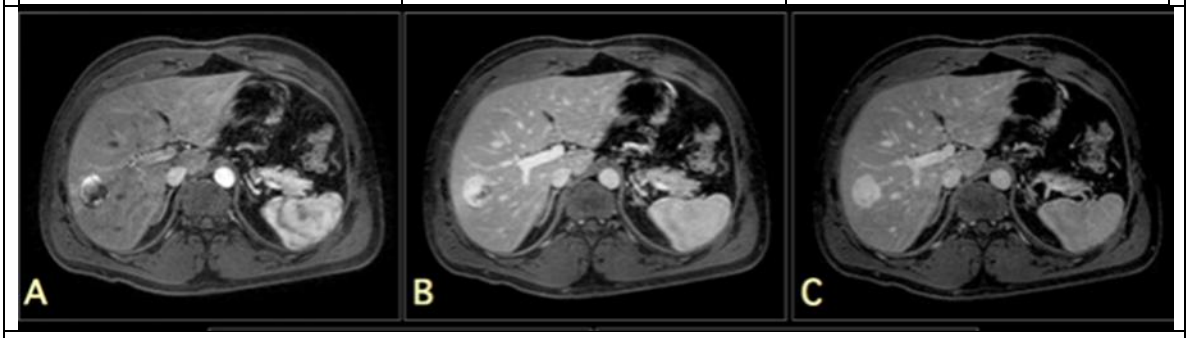
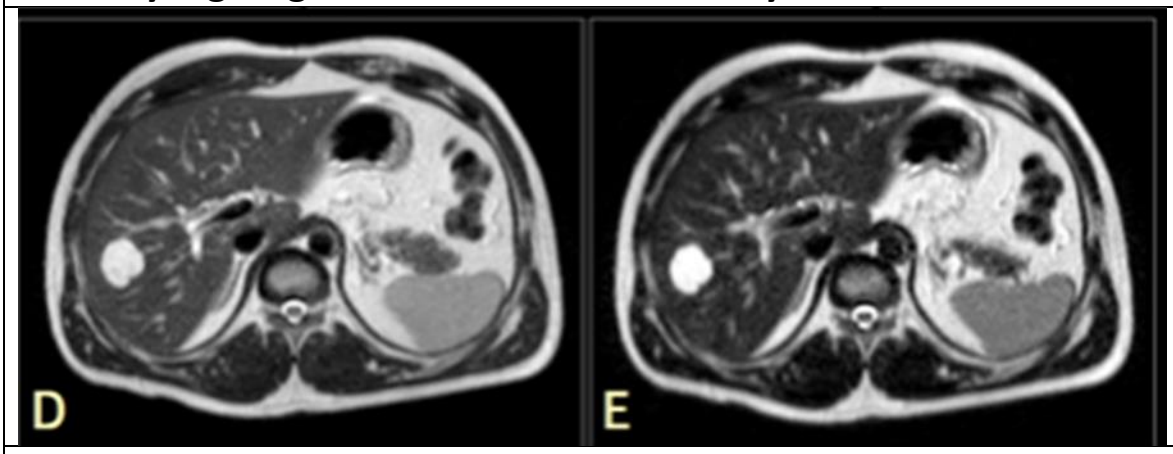
- Gd- BOPTA is generally not helpful for hemangiomas because they have variable appearance ranges from hypointensity to hyperintensity

T2	DWI
	

Giant Hemangioma



Small Hemangioma (pattern I)

Late arterial phase: Peripheral nodule enhancement	Portal venous phase: Centripetal fill in	Delayed phase: Complete homogenous enhancement
		
T2: Very high signal at moderate and heavy T2		
		

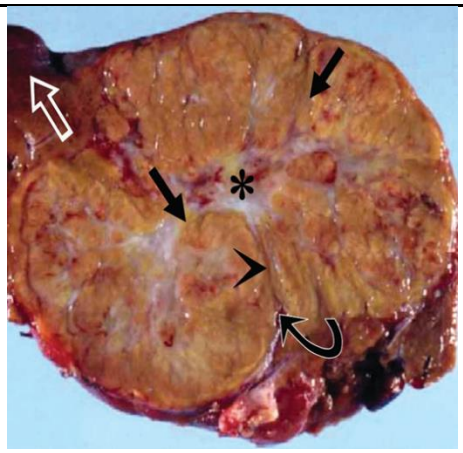
2- Focal Nodular Hyperplasia (FNH)

- 2nd most common benign liver tumor after hemangioma.
- 8% of all hepatic tumours
- 80%- 90% are solitary lesions <5cm in diameter
- Most common in middle aged females
- Not associated with any malignant potential
- Not associated with oral contraceptive pills.
- Enhancement of the central scar is pathognomonic for FNH.

- Age: Middle age.
- Sex: F >> M = 4:1

❖Pathological Classification

Type or Subtype
Classic FNH
Nonclassic FNH
Telangiectatic FNH
FNH with cytologic atypia
Mixed hyperplastic and adenomatous FNH

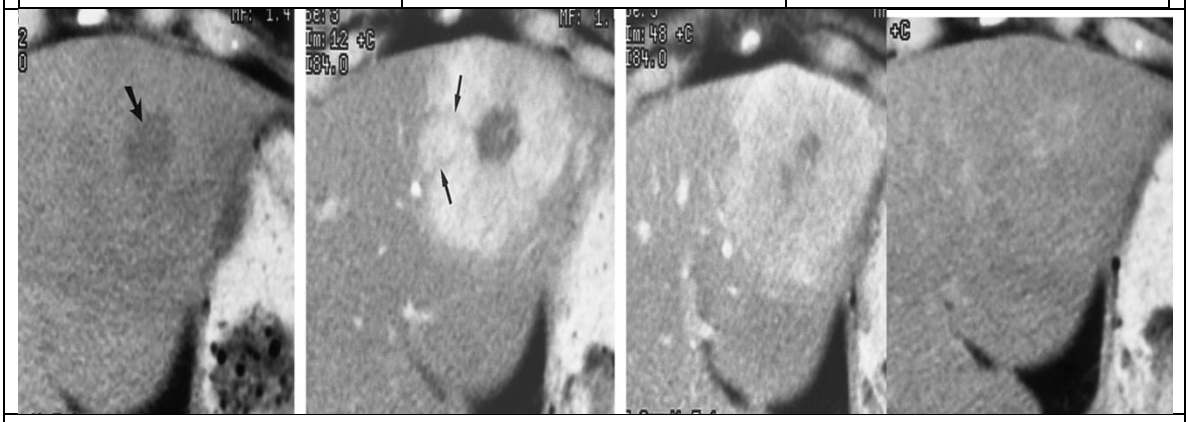


Imaging findings:

- Well defined lesion.
- Nodular surface.
- Pseudocapsule (high T2 signal and delayed contrast enhancment).
- ***Central scar (high T2 signal and delayed contrast enhancment).***
- Radiating septa
- Central feeding artery + Spoke-whell pattern.
- CT: isodense to liver.
- T1: iso or slightly hypo
- T2: iso or slightly hyper
- NO hge.
- NO intracellular fat.
- NO signal drop out on out of phase.

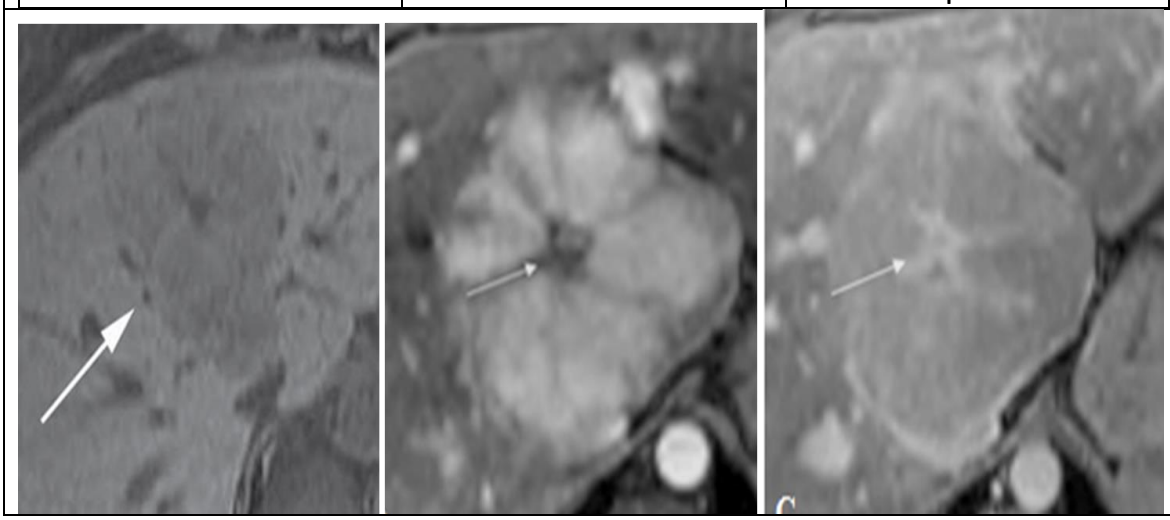
❖Triphasic CT

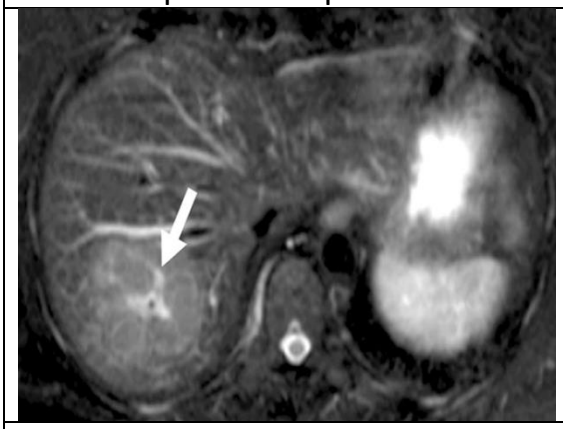
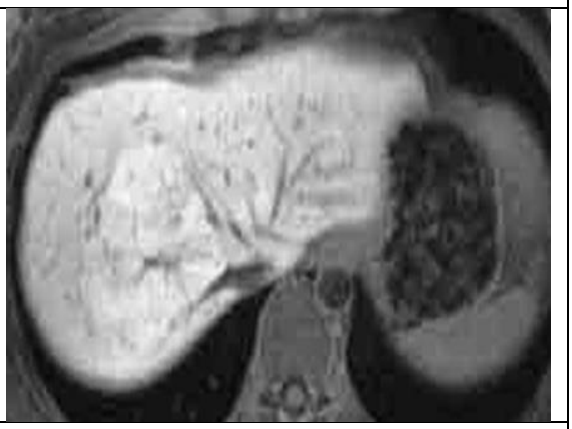
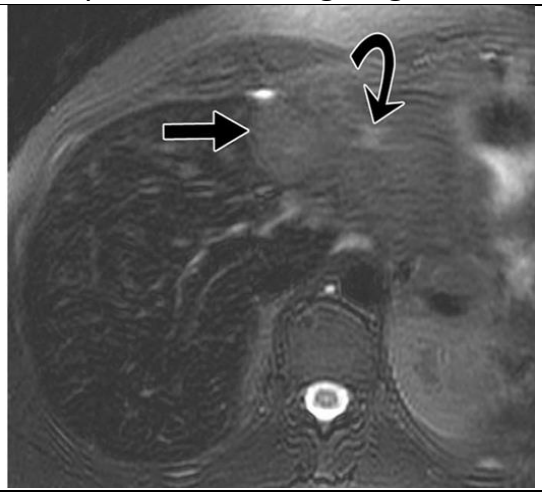
NCCT:	Arterial phase :	Delayed phase:
isodense or slightly hypodense ± low density central scar	Marked homogenous enhancement. + Hypodense central scar	Wash out of contrast + Slight enhancement of the central scar



❖Dynamic MRI

Precontrast phase:	Arterial phase:	Delayed phase:
iso or slightly hypointense ± hypointense central scar	Marked homogenous enhancement. + Hypointense central scar	Wash out of contrast + Slight enhancement of central scar + Pseudocapsule



T2 FS:	**Hepatic specific contrast agent Hepatobiliary phase:**
FNH: iso or slightly hyper High SI of: Central scar + Pseudocapsule + Septa	FNH iso- or slightly hyperintense. The central scar is hypointense.
	
SPIO specific contrast agent	
• The lesion and liver contain Kupffer cells => decreased signal • The central scar doesn't contain Kupffer cells => High signal	
	

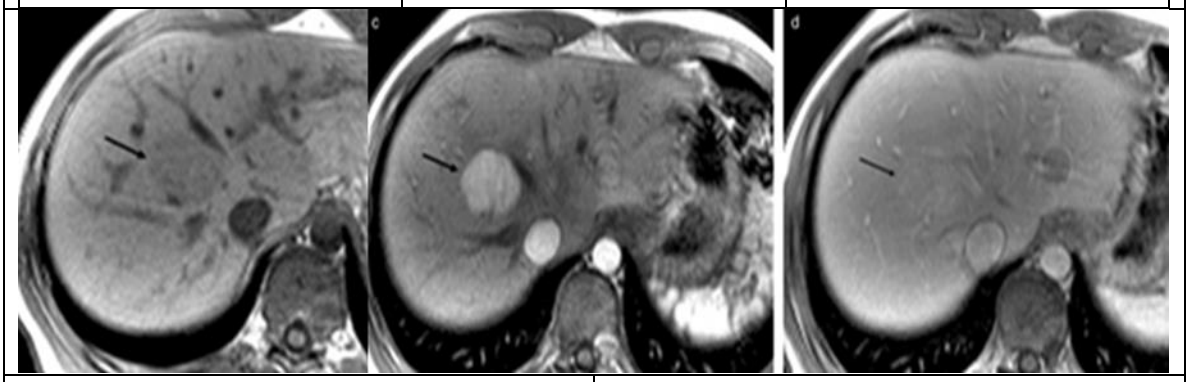
3- Hepatic Adenoma (HA)

- Rare benign tumors of the liver.
- Most commonly seen in women taking oral contraceptives.
- Solitary (80%)

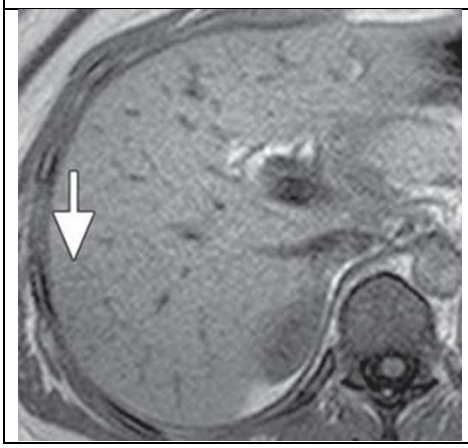
- Lesions > 5 cm => Intra-tumoral hemorrhage (with possible hepatic rupture)
- Classically, a hemorrhagic liver mass in a young woman taking oral contraceptives is highly suggestive of an adenoma.

❖Dynamic MRI

Pre-contrast:	**Arterial phase :**	**Delayed phase:**
Iso – slightly hypo	Marked homogenous enhancement.	Wash out of contrast

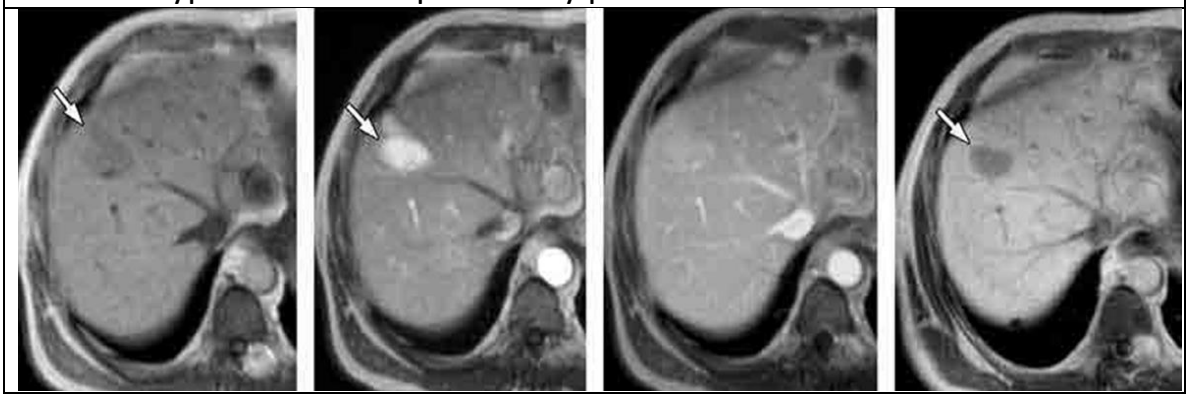


In of phase:	**Out of phase:**
Iso – slightly hypoitnese signal	Homogenous drop in signal intensity due to intra-lesional fat



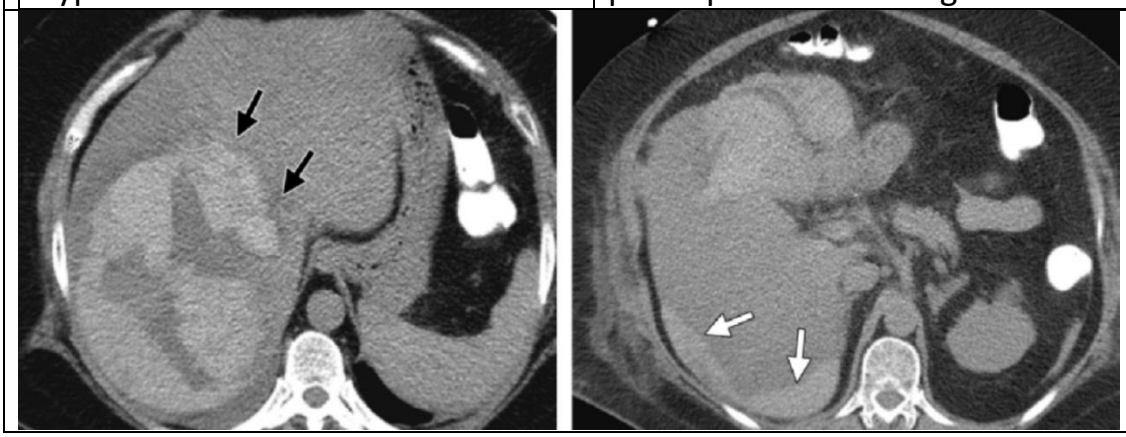
❖ Hepatic specific contrast agent (Gd-BOPTA)

- HA is hypointense in Hepatobiliary phase.



❖Ruptured Hepatic Adenoma

NCCT: Intra-tumoral hemorrhage = Hyperdense areas with tumor	NCCT: Tumor rupture with perihepatic hemorrhage



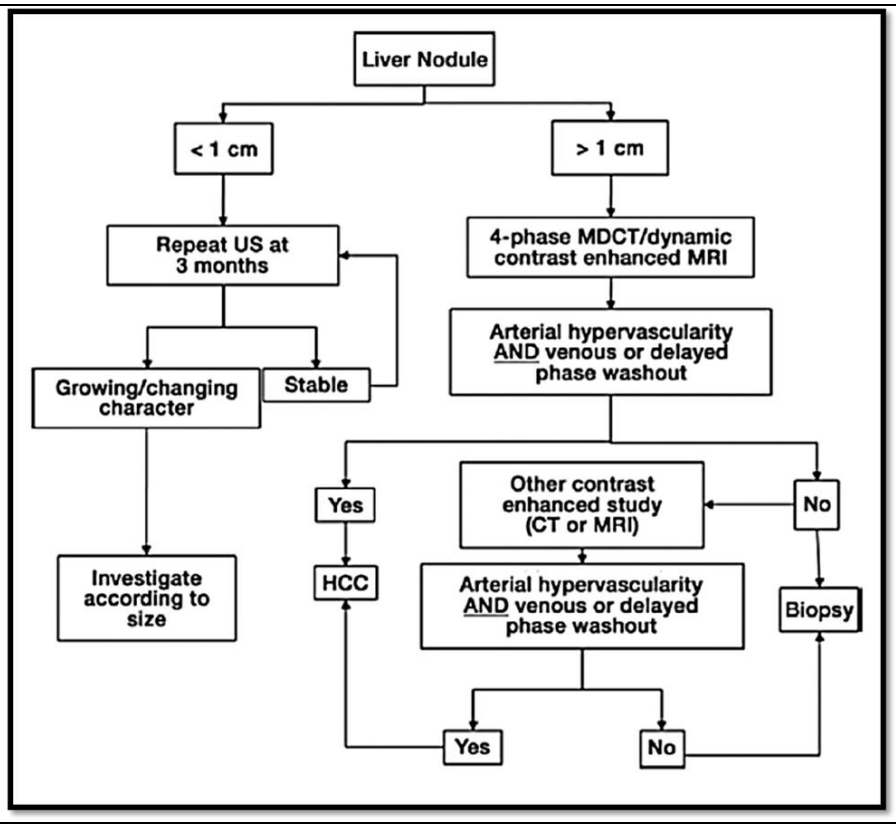
4- Hepatocellular Carcinoma (HCC)

- **6th most common cancer**
- **3rd most common cause of cancer-related death**

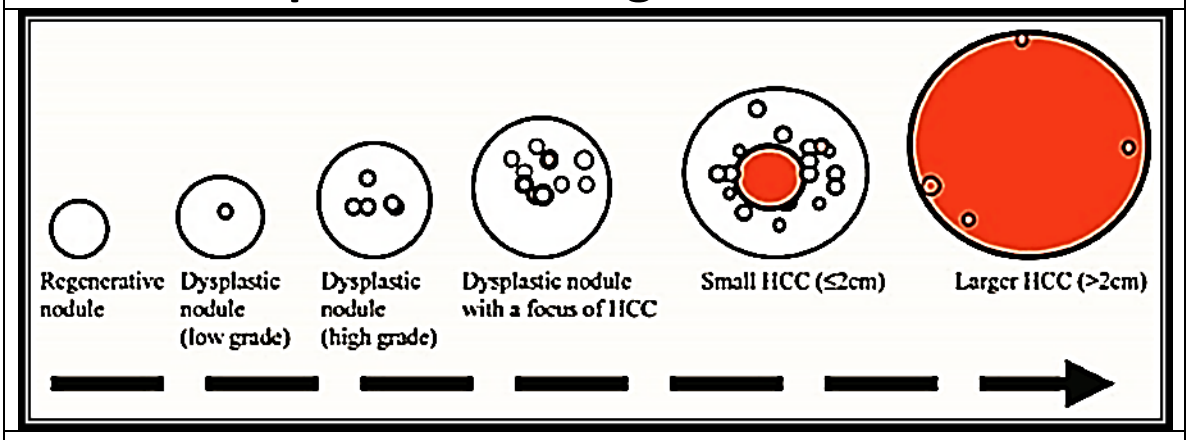
Risk Factors:

- Chronic viral hepatitis B/C
- Alcohol => Alcoholic steato-hepatitis (ASH)
- Non-alcoholic fatty liver diseases (NAFLD):
  - Steatosis => Nonalcoholic steato-hepatitis (NASH) => Cirrhosis
- Metabolic:
  - Hereditary haemochromatosis
  - Wilson disease
  - Alpha-1-antitrypsin deficiency

- **American Association for the Study of Liver Diseases AASLD Guidelines 2005, 2010**
- **US Surveillance for Cirrhotic Patients**



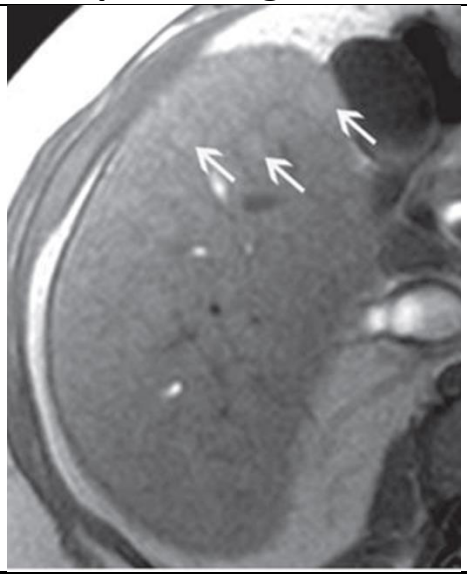
❖ Stepwise Carcinogenesis Of HCC



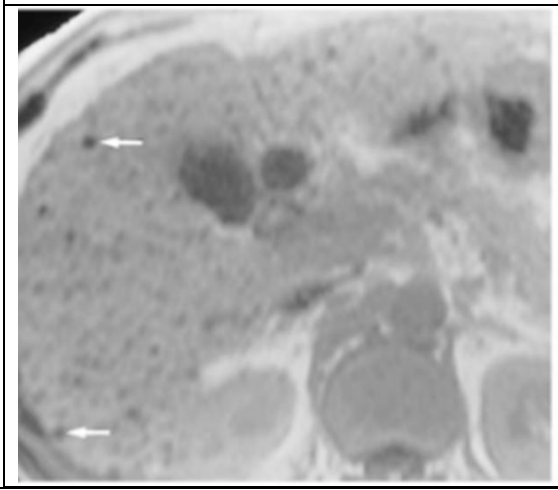
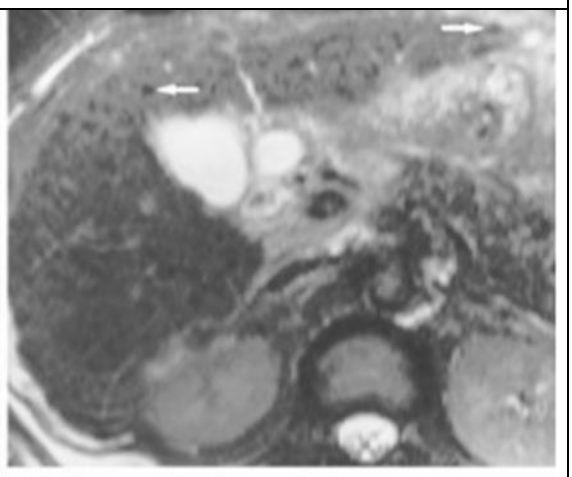
i) Regeneration Nodule (RN)

T1: variable SI nodules	T2 FS: No hyperintense nodules	T1 Arterial phase: No enhancing nodules

❖ Steatotic Regenerative Nodule

In of phase: High SI	Out of phase: Drop out of SI
	

❖ Siderotic Regenerative Nodule

T1: Low SI	T2 FS: Low SI
	

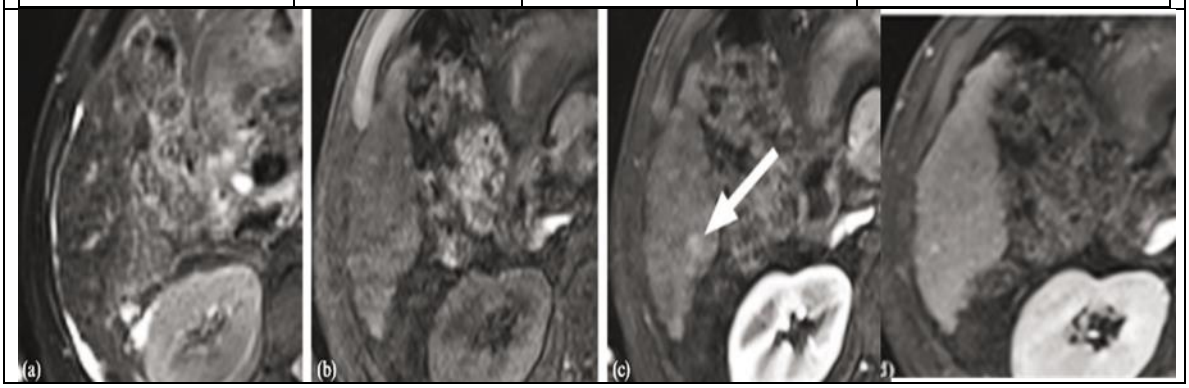
ii) Low Grade Dysplastic Nodule (LGDN)

<u>Radiologically appear similar to RN:</u>

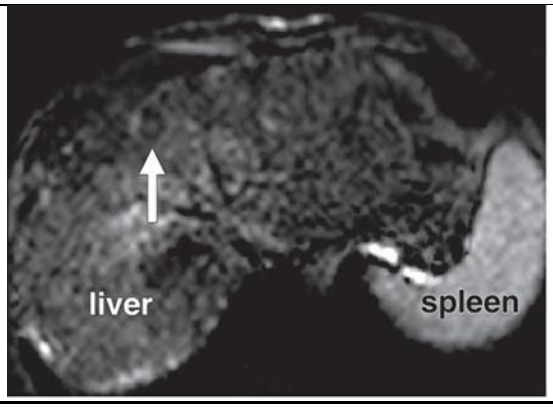
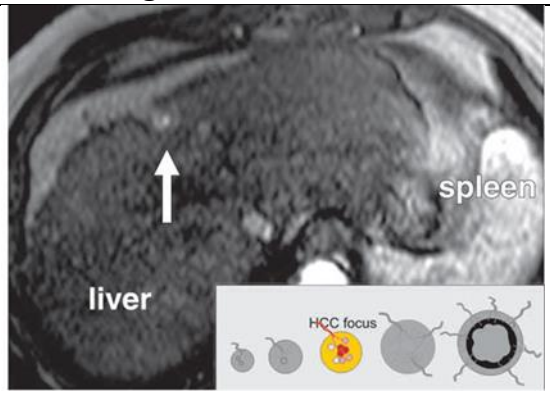
- T2: NO hyperintesne signal
- Arterial phase: NO enhancement
- SPIO enhanced MRI: Hypointense
- Gd-BOPTA hepatobiliary phase: Isointense to liver

iii) High Grade Dysplastic Nodule (HGDN)

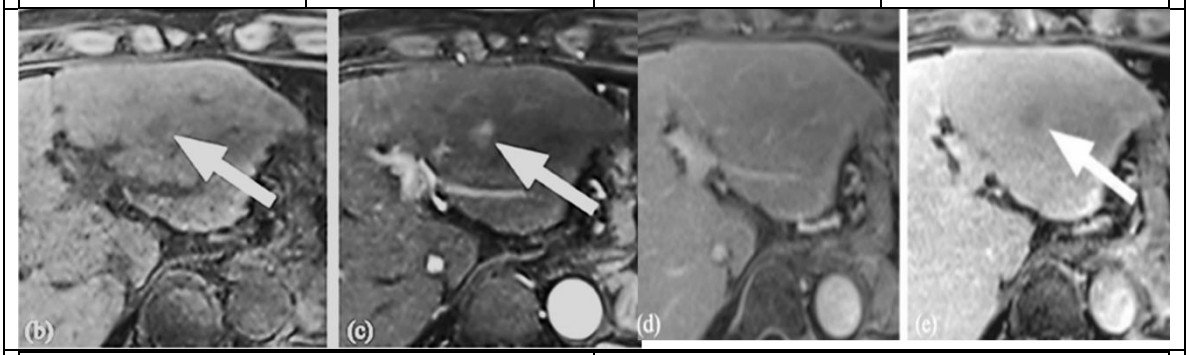
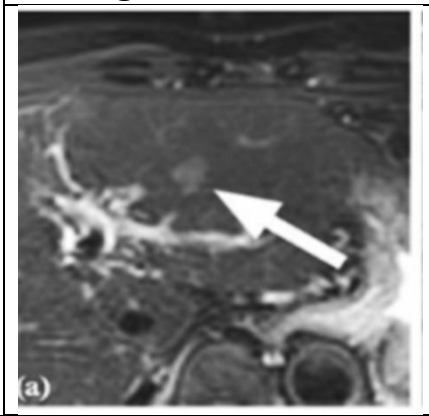
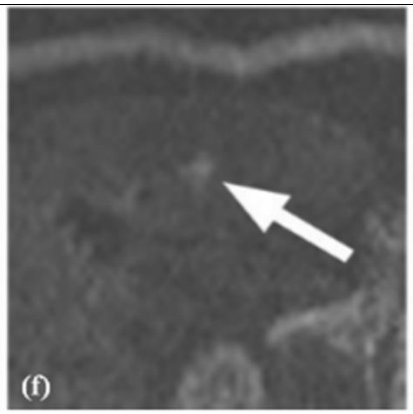
T2 FS	T1 pre	Arterial phase	Delayed phase
Iso intense		Faint enhancement	No wash out



iv) Dysplastic Nodule With HCC Focus

T2:	**Arterial phase:**
Nodule within nodule appearance	
High signal focus within low signal nodule	Enhancing focus within non-enhancing nodule
	

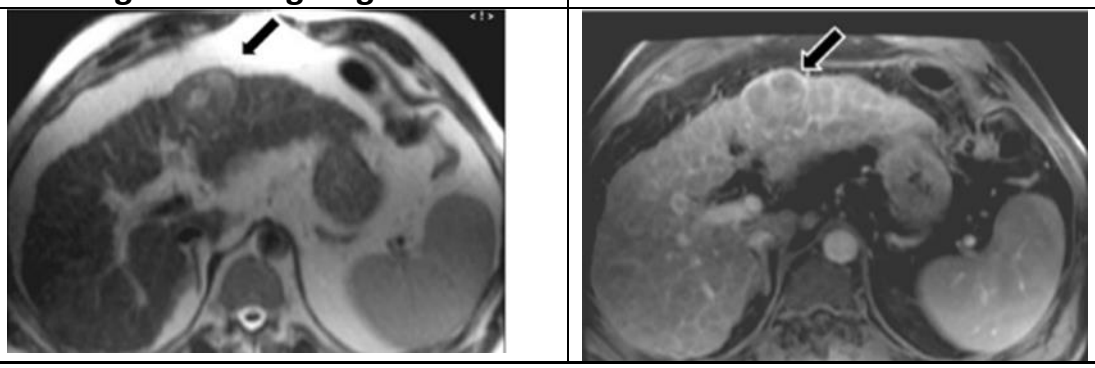
v) Small HCC <2cm

T1: Hypo	Arterial enhancement	Portal phase	Delayed wash out of contrast
			
T2: high SI		**DWI: Restricted**	
			

vi) Large HCC >2cm

T1: Low signal	Arterial enhancement	Portal phase	Delayed wash out of contrast

T2: Mosaic pattern = Heterogeneous High signal	**Delayed capsular enhancement**

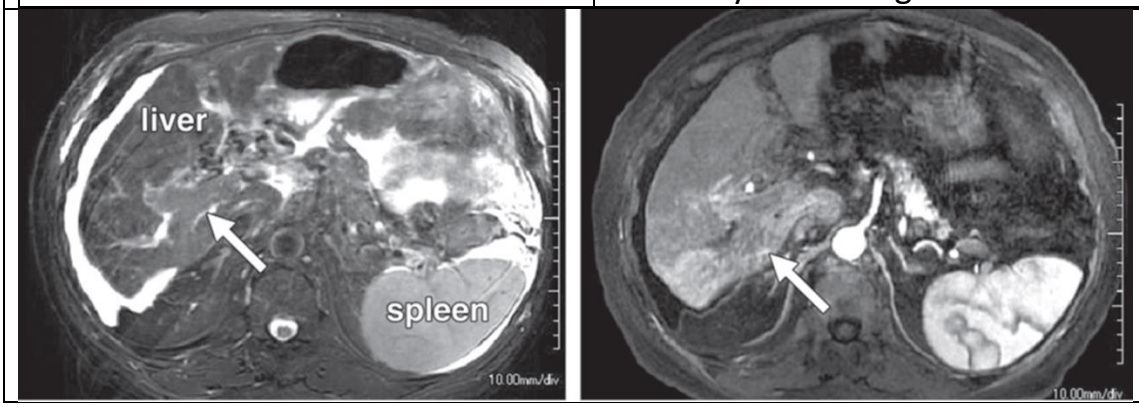


vii) Advanced HCC

- Extracapsular extension with formation of satellite nodules
- Vascular invasion: Venous Thrombosis.
- Extrahepatic dissemination.
- Regional lymphadenopathy
- Distant metastases

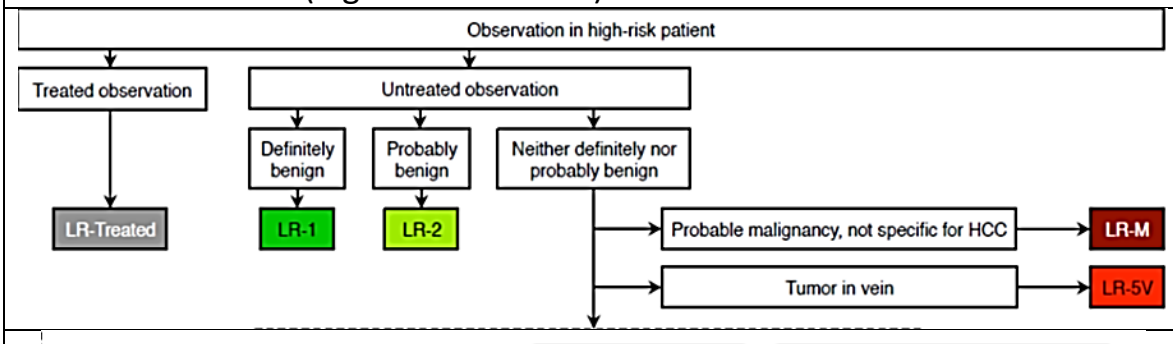
Right portal vein malignant thrombosis

	Arterially enhancing thrombus

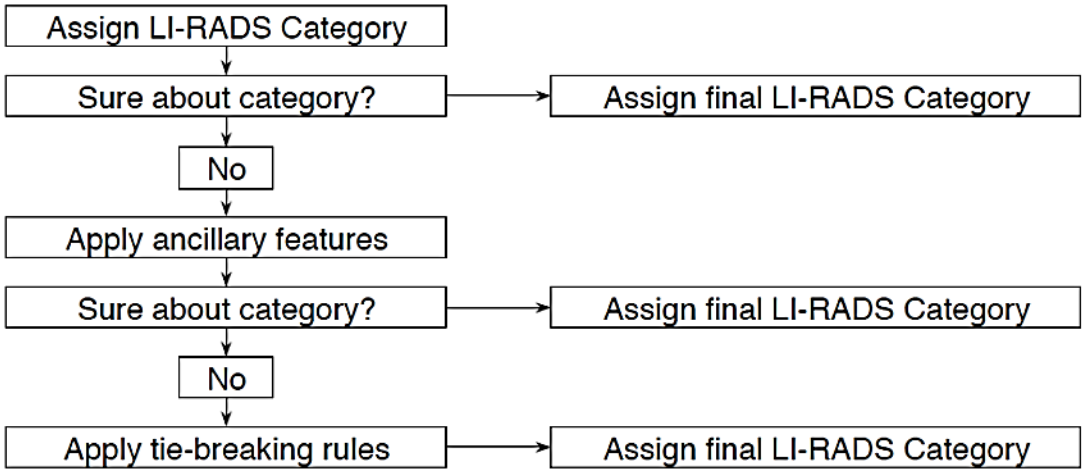


❖ LI-RADS (v2014)

- LI-RADS (Liver Imaging Reporting and Data System) is a set of standardised terminology and a classification system for imaging findings in liver lesions.
- LI-RADS score for a liver lesion is an indication of its relative risk for hepatocellular carcinoma (HCC).
- The classification system is meant to be used in livers which have risk factors for HCC (e.g. cirrhotic livers).



		Arterial phase hypo- or iso-enhancement		Arterial phase hyper-enhancement		
	Diameter (mm):	< 20	≥ 20	< 10	10-19	≥ 20
•"Washout" •"Capsule" •Threshold growth	None:	LR-3	LR-3	LR-3	LR-3	LR-4
	One:	LR-3	LR-4	LR-4	LR-4 / LR-5	LR-5
	≥ Two:	LR-4	LR-4	LR-4	LR-5	LR-5



A) Major Criteria

- Major features are imaging features used to categorize LR-3, LR-4, and LR-5

i. <u>Tumor diameter</u>

- Defined as the largest dimension of a lesion
- Measured from outer edge to outer edge in the imaging sequence, phase, and plane in which the margins are most sharply demarcated.
- Hypovascular lesions classified into 2 categories according to the size criteria (<2 cm vs. ≥2 cm)
- Hypervascular lesion classified into 3 categories according to the size criteria (<1 cm vs. 1-2 cm vs. ≥2 cm)

ii. <u>Arterial phase enhancement</u>

- Enhancement in the arterial phase is definitely greater than that of background liver
- if unsure, classify as isoenhancing

iii. <u>Portal venous and delayed Washout</u>

- Relative hypointensity of the lesion compared with background liver on the portal venous and delayed phases

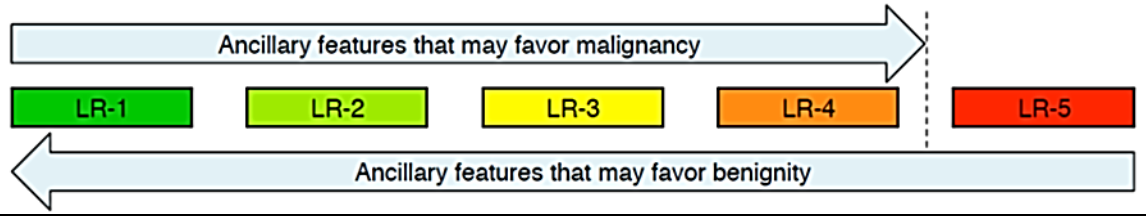
iv. <u>Portal venous and delayed Capsule/pseudocapsule</u>

- Peripheral rim of smooth hyperenhancement seen on the portal venous or delayed phases

v. <u>Threshold growth</u>

- Diameter increase of a mass by a minimum of 5 mm
- if prior exam ≤6 months, diameter ≥50% increase
- if prior exam >6 months, diameter ≥100% increase
- A new 10 mm lesion represents threshold growth, regardless of time interval
- Threshold growth only applies to masses
- Threshold growth should be compared on similar sequences between studies

B) Ancillary Features:



i. Ancillary Features favoring HCC

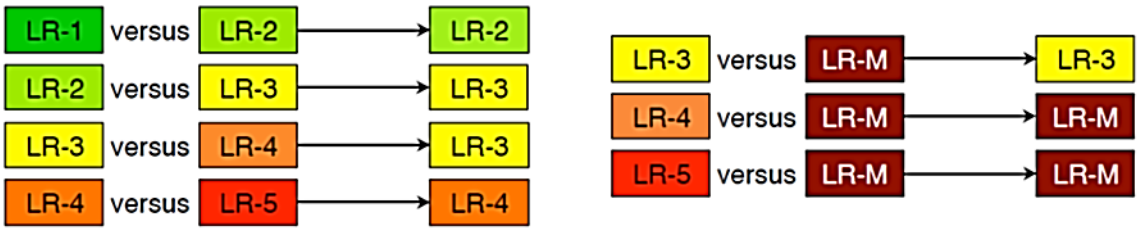
- Mild-moderate T2 hyperintensity
- Restricted diffusion
- Corona enhancement (rim of peri-lesional enhancement)
- Mosaic architecture
- Nodule-in-nodule architecture
- Intralesional fat
- Diameter increase less than threshold growth!
- Lesional iron sparing
- Lesional fat sparing
- Blood products
- Distinctive rim
- Hepatobiliary phase hypo-intense rim
- Hepatobiliary phase hypo-intensity

ii. Ancillary Features favoring Benignity

- Undistorted vessels
- Homogeneous marked T2 hyperintensity
- Homogeneous marked T2 or T2* hypointensity
- Parallels blood pool enhancement
- Diameter reduction
- Diameter stability ≥ 2 years
- Hepatobiliary phase iso-intensity

C) Tie-breaking Rules:

- If, after application of ancillary features, a radiologist is still unsure about the final category for an observation, tie-breaking rules should be applied.
- The tie-breaking rules move observations to a category with a lower degree of certainty.


LR-1 versus LR-2 → LR-2
LR-2 versus LR-3 → LR-3
LR-3 versus LR-4 → LR-3
LR-4 versus LR-5 → LR-4
LR-3 versus LR-M → LR-3
LR-4 versus LR-M → LR-M
LR-5 versus LR-M → LR-M

❖LIRADS Categories

LR-1 **Definitely Benign**	**Concept:** 100% certainty observation is benign. **Definition:** Observation with imaging features diagnostic of a benign entity, or definite disappearance at follow up in absence of treatment.
LR-2 **Probably Benign**	**Concept:** High probability observation is benign. **Definition:** Observation with imaging features suggestive but not diagnostic of a benign entity.
LR-3 **Intermediate probability for HCC**	**Concept:** Both HCC and benign entity have moderate probability. **Definition:** Observation that does not meet criteria for other LI-RADS categories.
LR-4 **Probably HCC**	**Concept:** High probability observation is HCC but there is not 100% certainty. **Definition:** Observation with imaging features suggestive but not diagnostic of HCC.
LR-5 **Definitely HCC**	**Concept:** 100% certainty observation is HCC. **Definition:** Observation with imaging features diagnostic of HCC or proven to be HCC at histology.
LR-5V **Definitely HCC with Tumor In Vein**	**Concept:** 100% certainty that observation is HCC invading vein. **Definition:** Observation with imaging features diagnostic of HCC invading vein.
LR-M **Probably Malignant, not specific for HCC**	**Concept:** Observation is probably malignant, but imaging features are not specific for HCC. **Definition:** Observation with imaging features suggestive of non-HCC malignancy.
LR-Treated **Treated Observation**	**Concept:** A loco-regionally treated observation. **Definition:** Observation of any category that has undergone loco-regional treatment.

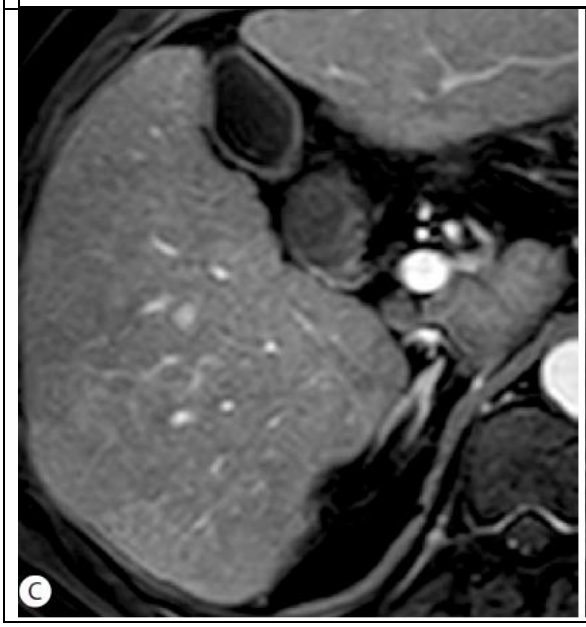
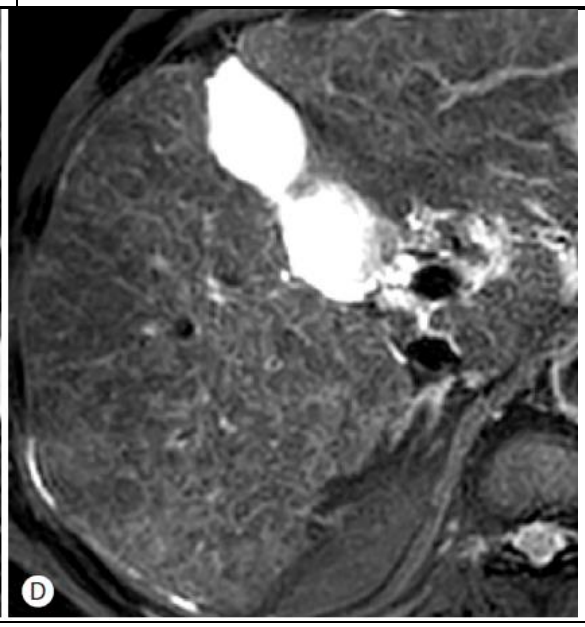
LR-1 (100% Benign)
⇨ Continued routine surveillance

- Cyst
- Haemangioma
- Vascular anomaly
- Perfusion alteration
- Hypertrophic pseudomass
- Confluent hepatic fibrosis
- Focal scar
- Definite disappearance at follow up in the absence of treatment

LR-2 (Probably Benign)
⇨ Continued routine surveillance

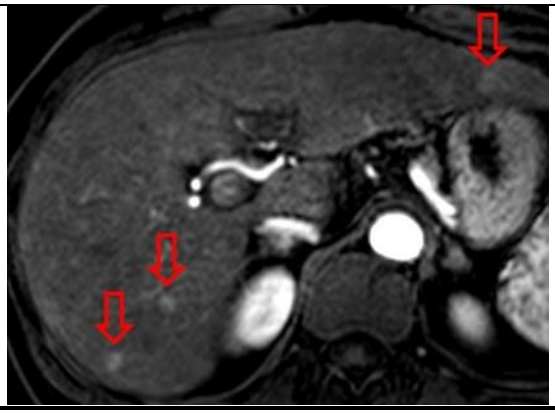
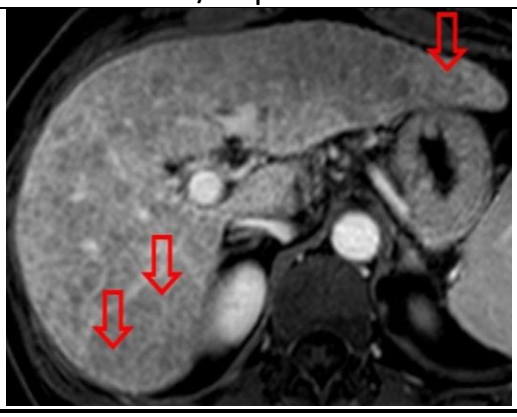
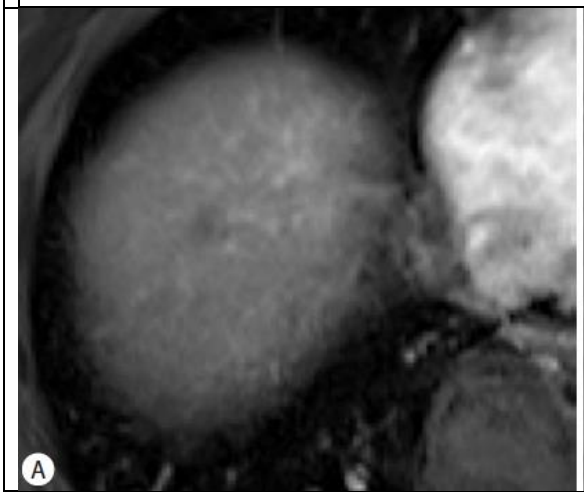
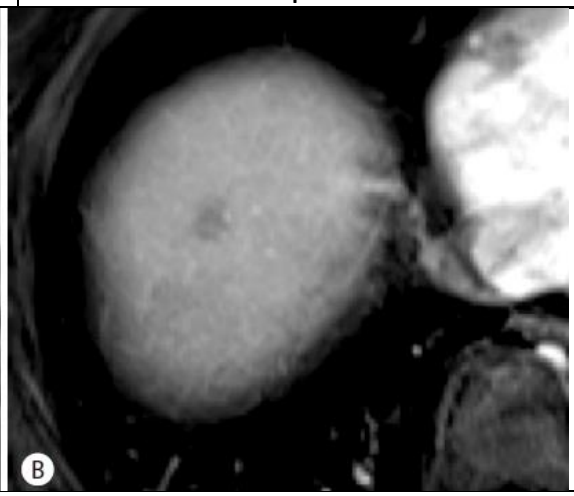
- **<u>Atypical appearance of:</u>**
    - Cyst
    - Haemangioma
    - Vascular anomaly
    - Perfusion alteration
    - Hypertrophic pseudomass
    - Confluent hepatic fibrosis
    - Focal scar
- **<u>LR-2 cirrhosis-associated nodule:</u>**
    - Diameter < 20mm AND
    - Homogeneous AND
    - Iso-enhancement to background cirrhotic nodules in all phases AND
    - Differ from background nodules (≥ 1 of following):
        - Distinctly larger than background nodules (but still < 20mm)
        - Mild to moderate CT hyper-attenuation
        - Mild to moderate T1 hyper-intensity
        - Mild T2 or T2* hypo-intensity
        - Moderate or marked T2 or T2* hypo-intensity

LR-2 cirrhosis-associated nodule:

T1 hyperintense nodule <2cm	No arterial enhancement
No washout	**T2 isointense**
	

LR-3 (Intermediate Probability for HCC)

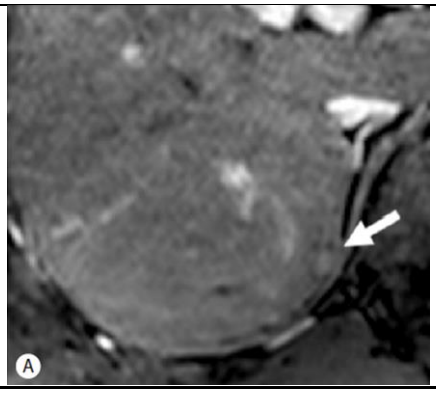
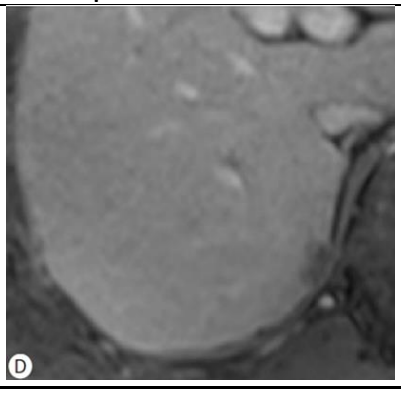
- Not a definitely benign entity, but not definitely HCC
- Mass with hepatic arterial phase iso- or hypoenhancement
  - <20 mm with no more than one of the following:
    - "washout"
    - capsule
    - threshold growth
  - ≥20 mm with no "washout," capsule, or threshold growth
- Mass with hepatic arterial phase hyperenhancement
  - <20 mm with no "washout," capsule, or threshold growth

Arterial enhancement Nodules < 2cm	Portal venous and delayed phase No washout / capsule
	
Arterial phase hypo Nodule < 2cm	Portal venous and delayed phase Washout + No capsule
	

LR-4 (Probably HCC)

⇨ Close follow up, additional imaging, biopsy, or treatment

- **<u>Mass with hepatic arterial phase iso- or hypoenhancement</u>**
  - <2cm + two or more of the following
  - >2cm + one or more of the following
    - "washout"
    - capsule
    - threshold growth
- **<u>Mass with hepatic arterial phase hyperenhancement</u>**
  - <10 mm + one or more of the following:
    - "washout"
    - capsule
    - threshold growth
  - 10-19 mm + only one of the following:
    - "washout"
    - (if just washout and was seen as a discrete nodule on a prior screening ultrasound, then "LR-5us" us =ultrasound)
    - capsule
    - threshold growth
    - (if just threshold growth, then "LR-5g" g=growth)
  - ≥ 20mm + none of the following
    - "washout"
    - capsule
    - threshold growth

Arterial enhancement Nodule size = 11-19mm	Delayed washout No capsule
	

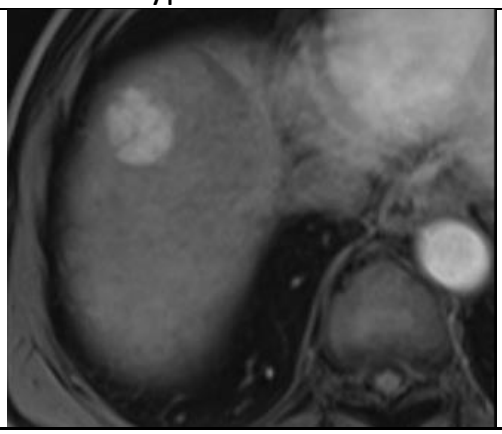
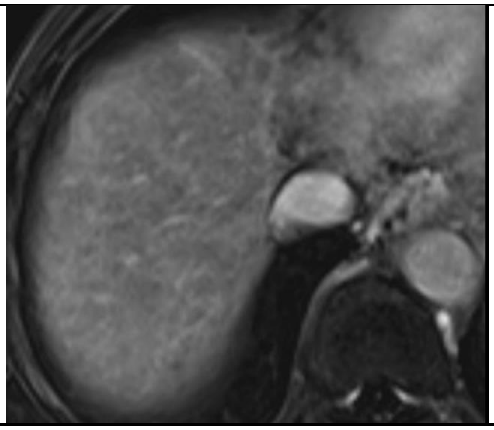
→ if it detected at US: LR-4 => LR-5us

→ if showed ≥50% diameter increase in ≤6 months: LR-4 => LR-5g

LR-5 (100% definite HCC)

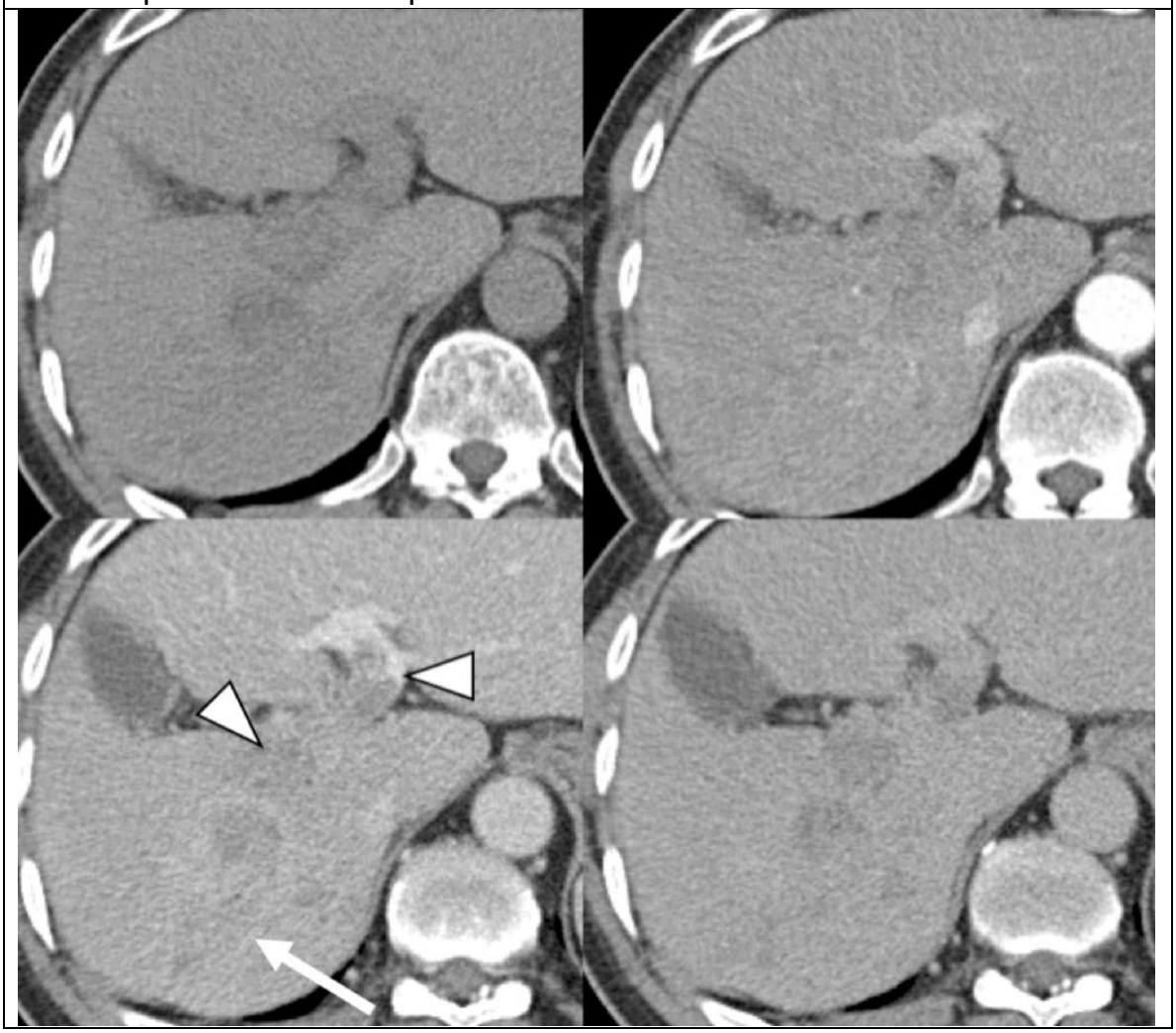
⇨ Treatment without biopsy, radiologic T-staging

- Mass with hepatic arterial phase hyperenhancement
  - Size = 10-19 mm mass + two or more of the following
  - Size >20 mm mass + one or more of the following
    - "washout"
    - capsule
    - threshold growth

Nodule 10-19mm + Arterial hyperenhancement	Delayed washout + Capsular enhancement
Nodule > 2cm Arterial hyperenhacement	Delayed washout
	

LR-5V

- LR-5V category is assigned when there is definite enhancing soft tissue in a vein irrespective of the presence or absence of visible intraparenchymal HCC.
- This category was created because macrovascular invasion usually constitutes a contraindication to curative treatments such as liver transplantation and hepatic resection.



LR-M

- LR-M category is assigned to malignant tumor but not specific for HCC.

- Intrahepatic cholangiocarcinoma (CC)
- combined HCC-CC
- Metastasis
- Lymphoma
- Post-transplant lymphoproliferative disease

LR-T

- LR-Treated category is assigned to treated observations, and is currently under development.
- Criteria for assessment of residual or recurrent disease based on the type of therapy will be released in future versions of LI-RADS.

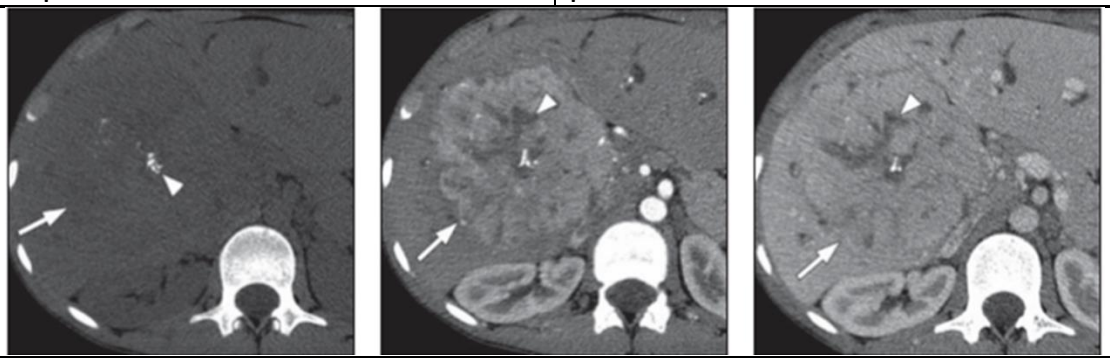
5- Fibrolamellar Hepatocellular Carcinoma

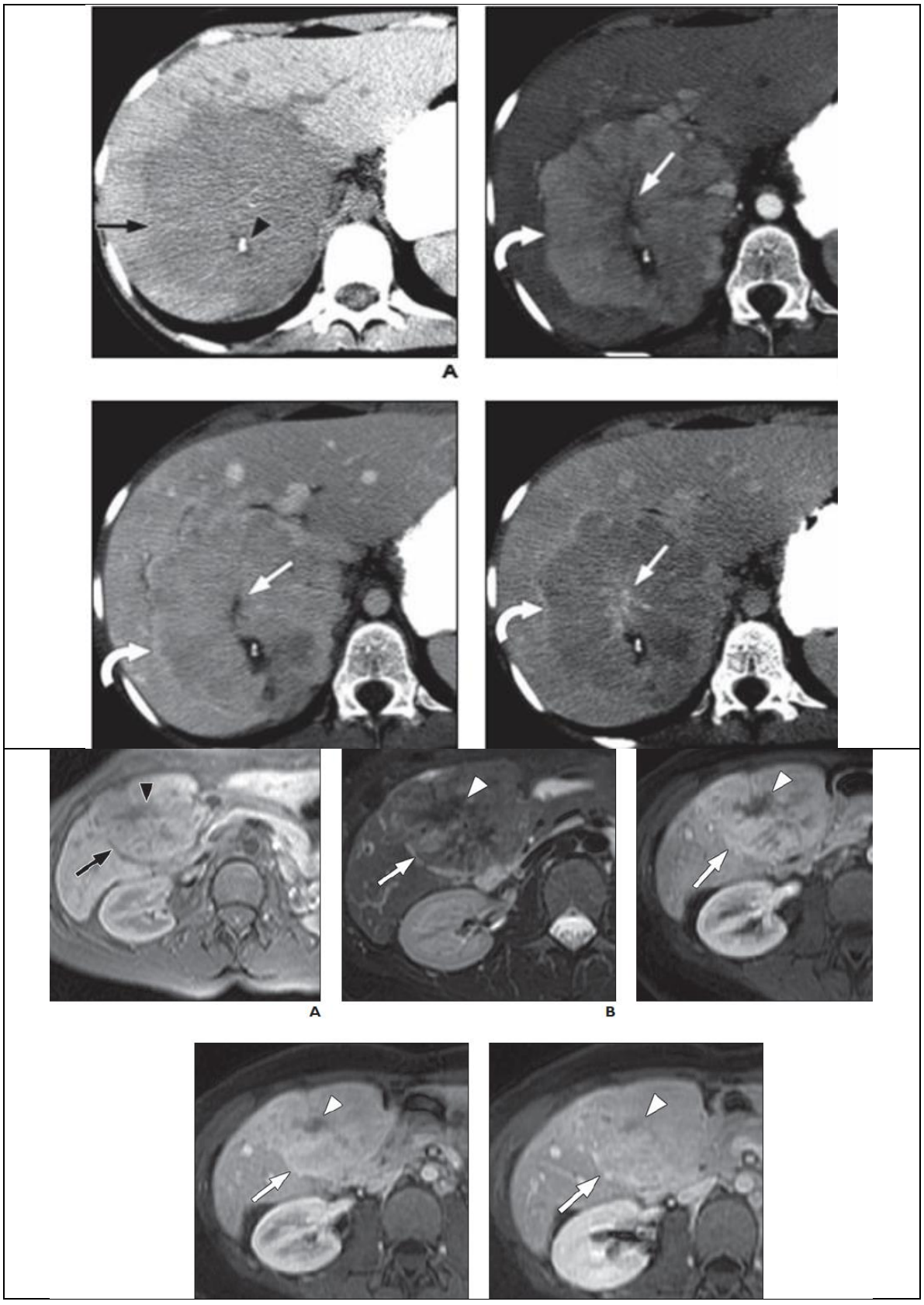
- Fibrolamellar HCC is an uncommon type of HCC.

<u>5 distinct aspects compared to generic HCC are:</u>

- Young adults (20 to 40 years of age)
- Do not have an association with cirrhosis, alcoholism or hepatitis B/C
- Not associated with raises serum alpha feto protein
- Usually large at presentation
- Less aggressive with a better 5-year survival

Fibrolamellar HCC	FNH
• Large, well-circumscribed focal lesion	
• ***A central radiating scar:*** ***T2 Hypointense***	• ***A central radiating scar:*** ***T2 Hyperintense***
• ***Calcifications*** within the central scar (35-65% of cases)	• ***Calcification is rare***
T1: iso to hypointense to the liver	
T2: iso to hyperintense to the liver	
Arterial phase: heterogeneous enhancement	
Portal venous / delayed phase: iso to hypointense	
Central scar may show delayed enhancement Should not be used as a differentiating criterion between FNH and fibrolamellar HCC	
• **<u>Hepatic specific contrast agent:</u>** hypointense in hepatobiliary phase	• **<u>Hepatic specific contrast agent:</u>** iso or hyperintense in hepatobiliary phase




A
A
B

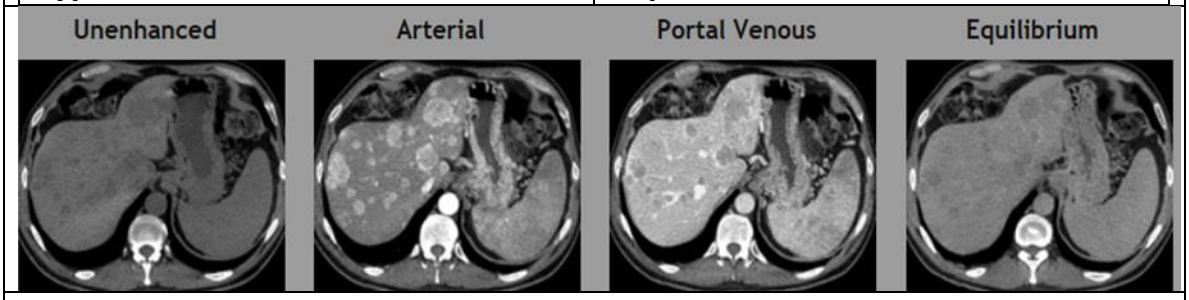
6- Liver Metastasis

i) Hypervascular Metastasis:

- These lesions draw their blood supply from the hepatic artery.
- These lesions will show marked enhancement during the arterial phase with delayed peripheral or central washout in the portal venous and equilibrium phases.

- **Carcinoid tumor**
- **Renal cell carcinoma**
- **Thyroid carcinoma**
- **Choriocarcinoma**
- **Breast carcinoma**
- **Melanoma**
- **Islet cell tumor**

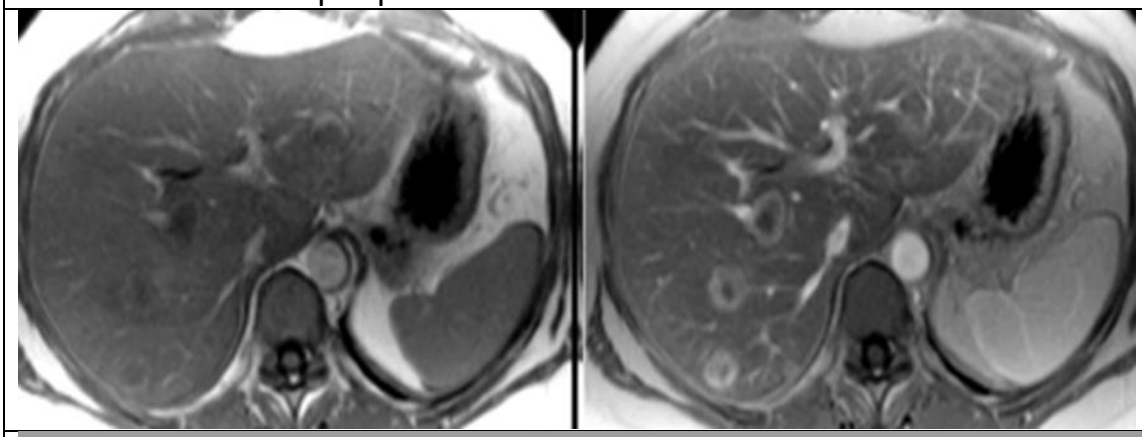
Arterial phase multiple hypervascular metastases	**Portal venous phase: Rapid washout of contrast**

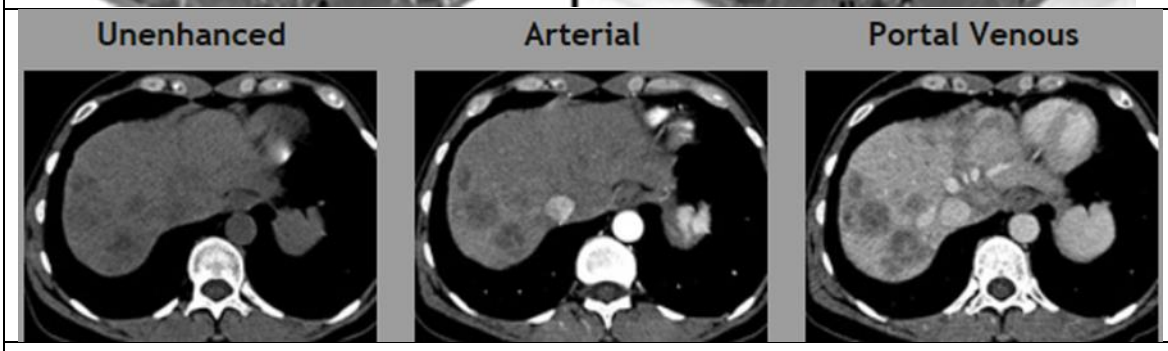


Hypervascular Metastasis with central necrosis

ii) Hypovascular Metastases

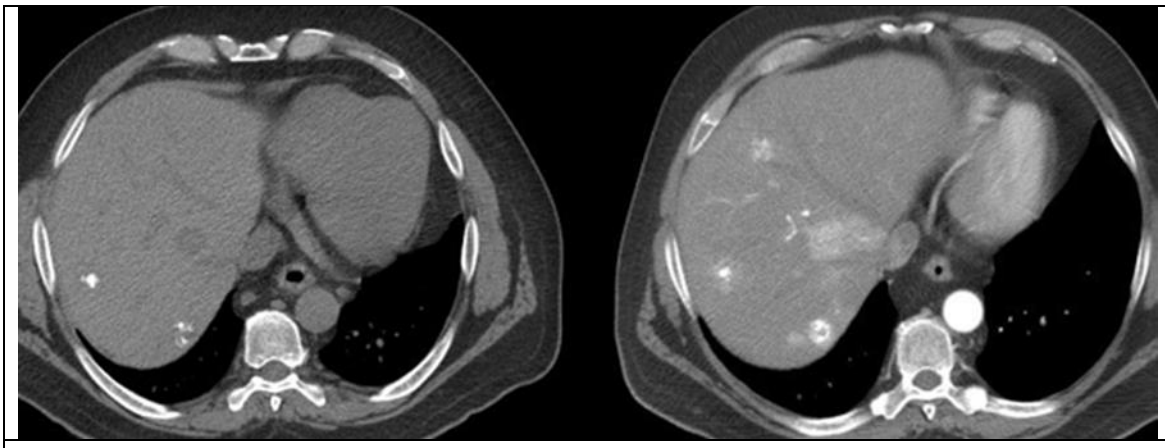
- Hypovascular metastases show decreased enhancement relative to normal liver and are most conspicuous on portal venous phase images.
- Most common and occur in GI tract, lung, breast and head/neck tumors.
- They are detected as hypodense lesions in the late portal venous phase sometimes with peripheral enhancement.





iii) Calcified Metastases

- Mucinous neoplasms:
  - Colon is the most common
  - GIT
- Serous ovarian cystadenocarcinoma
- Medullary thyroid carcinom.
- Sarcomas: Osteosarcoma, leiomyosarcoma.
- Islet cell carcinoma of the pancreas
- Breast carcinoma



iv) Cystic Metastases

- Mucinous ovarian ca
- Colon ca
- Sarcoma
- Melanoma
- Lung ca
- Carcinoid tumor.

v) Most Common Adult Hepatic Metastasis

- Colon
- Stomach
- Pancreas
- Breast
- Lung
- Eye

vi) Most Common Pediatric Hepatic Metastasis

- Neuroblastoma
- Wilms tumor
- Leukemia

B- Pediatric Solid Hepatic Focal Lesions

1- Hepatoblastoma

- 1st common primary hepatic tumor in children.
- A malignant neoplasm whose cells resemble embryonic liver

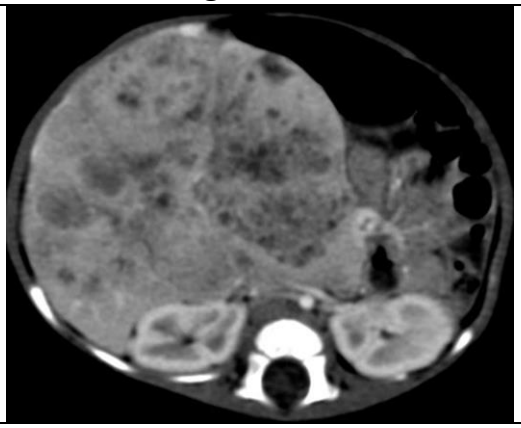
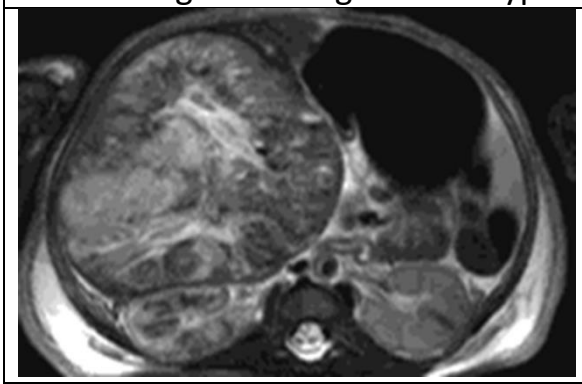
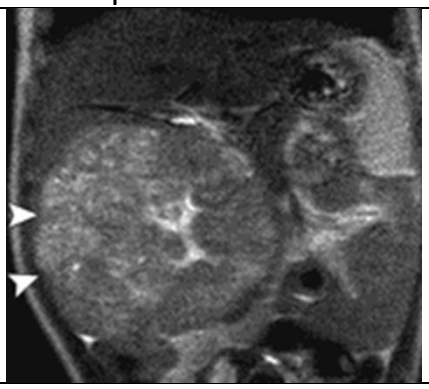
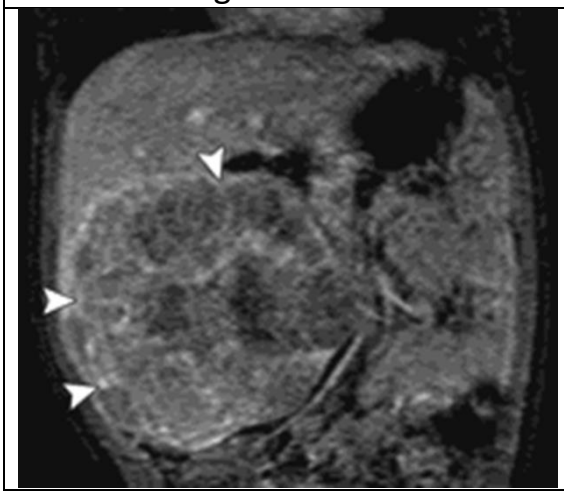
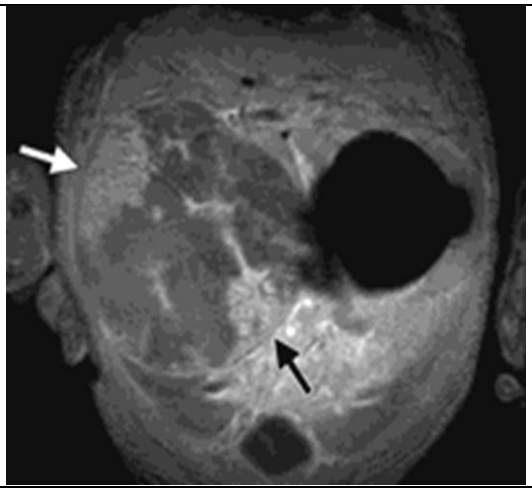
- Age: < 5 years of age
- 90% show abnormal elevation of AFP level

Associations:

- Beckwith-wiedemann syndrome
- Gardner syndrome
- Familial adenomatous polyposis
- Type 1A glycogen storage disease
- Trisomy 18.

Imaging findings:

- A well defined hepatic mass
- Heterogeneous: areas of necrosis and haemorrhage.
- Chunky, dense calcifications (40% of cases)
- T1: generally hypo intense
- T2: hyper intense compared to liver
- +C: heterogeneous enhancement less than normal liver parenchyma.
- Like HCC, hepatoblastoma can invade the portal or hepatic veins

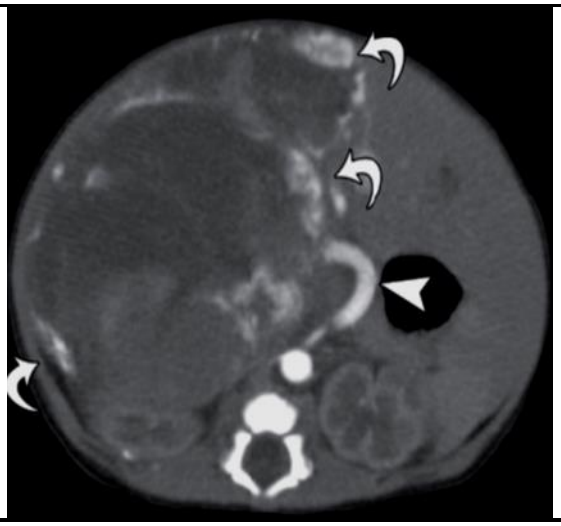
NCCT: Calcifications	CECT: heterogeneous enhancement
	
T2: heterogeneous signal with hypointense septa in between	
	
T1+C: heterogeneous enhancement	
	

2- Infantile Hemangioendothelioma

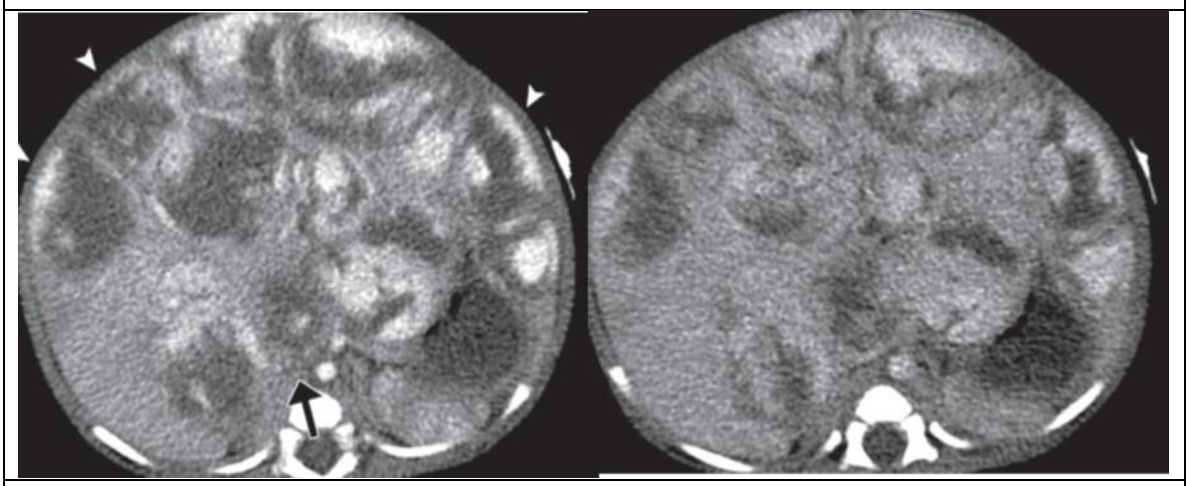
- 1st most common benign hepatic tumor of infancy.
- Benign vascular tumor composed of vascular channels lined by endothelial cells.
- ½ Solitaty ½ Multifocal

Imaging findings:

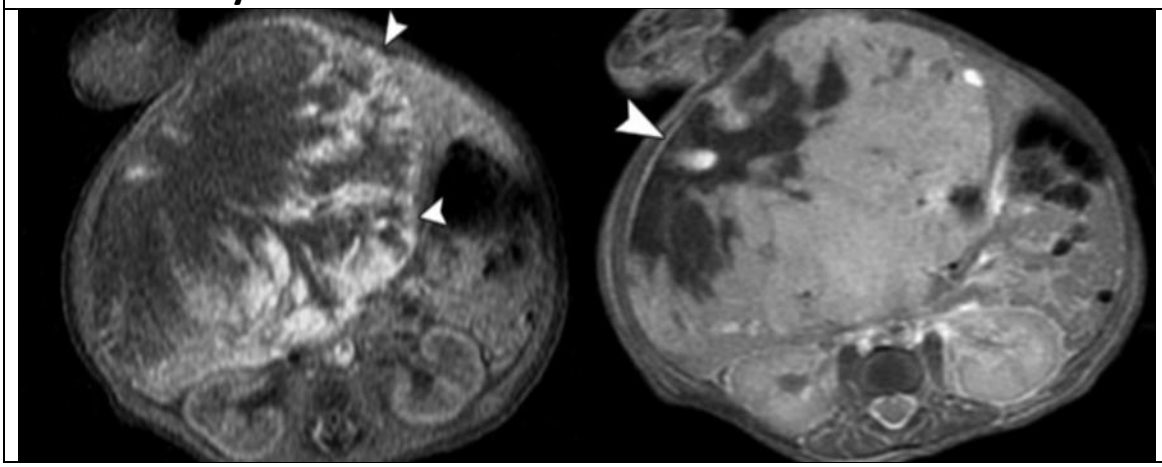
- Enlarged celiac trunk and hepatic artery
- Reduction in the aortic caliber (mid-aortic syndrome) below the level of coeliac branch because of the important vascular distribution toward the liver
- T1: hypointense
- T2: hyperintense
- Arterial phase: intense, peripheral nodular enhancement
- Portal and venous phase: progressive centripetal filling



❖ **Multifocal form**

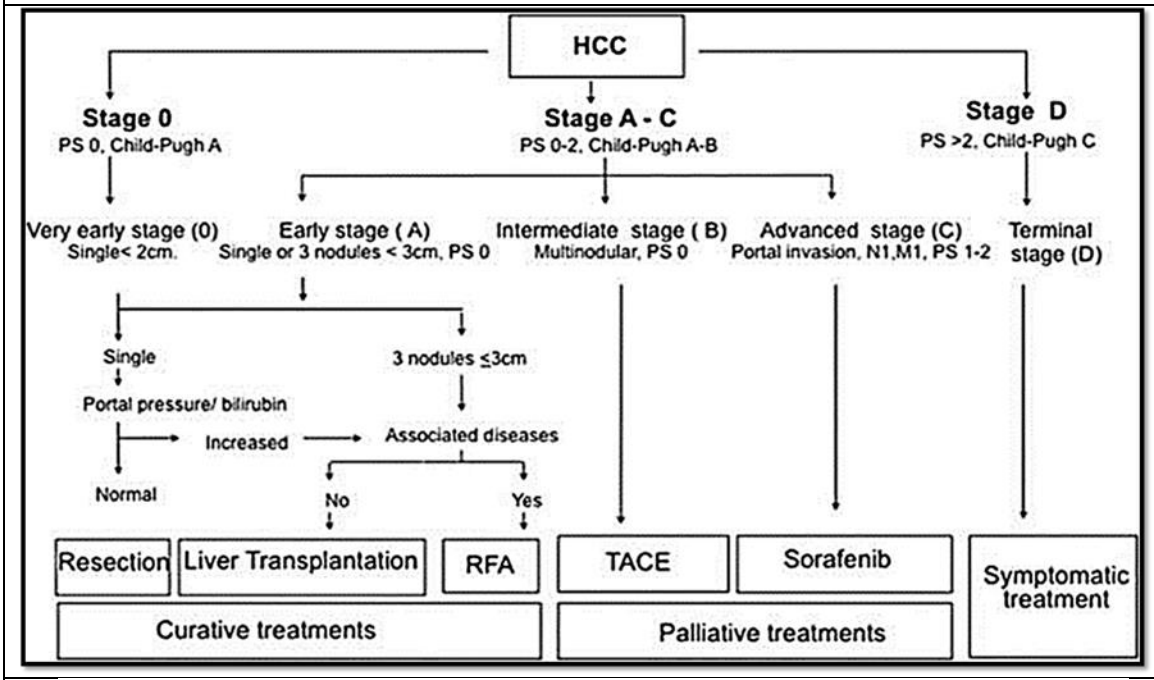


❖ **Solitary form**



Liver Tumor Staging

Barcelona Clinic Liver Cancer BCLC Staging



BCLC	Tumor stage	C-P	PS	TTT
Stage 0	Solitary <2cm	A	0	Resection Transplantation RFA
Stage A	Solitary <5cm 3 Nodules<3cm	A or B	0	Resection Transplantation RFA
Stage B	**Multinodular** No >3nodules or Size>3cm	A or B	0-2	TACE
Stage C	Vascular invasion Metastasis N1 M1	A or B	0-2	Sorafenib
Stage D	Any	C	3-4	Supportive

❖ Child-Pugh Classification

Parameter	Points assigned		
	1	2	3
Ascites	Absent	Slight	Moderate
Hepatic encephalopathy	None	Grade 1-2	Grade 3-4
Bilirubin micromol/L (mg/dL)	<34.2 (<2)	34.2-51.3 (2-3)	>51.3 (>3)
Albumin g/L (g/dL)	>35 (>3.5)	28-35 (2.8-3.5)	<28 (<2.8)
Prothrombin time Seconds over control INR	 <4 <1.7	 4-6 1.7-2.3	 >6 >2.3
CPT classification: Child A: score 5-6 (well compensated); Child B: score 7-9 (significant functional compromise); Child C: score 10-15 (decompensated)			

❖ Eastern Cooperative Oncology Group (ECOG) Scheme

ECOG System for Scoring Patient Performance Status

Score	Characterization
0	Fully active
1	Unable to pursue strenuous physical exercise
2	Symptomatic but able to completely fulfill normal activities of daily living
3	Limited ability to perform activities of daily living, spends more than half the day in bed
4	Completely bedridden

❖TNM Staging

TABLE 1
TNM Staging System for Liver Tumors

Stage	Definition
Primary Tumor	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
T1	Solitary tumor ≤2 cm without vascular invasion
T2	Solitary tumor ≤2 cm with vascular invasion, or multiple tumors in one lobe, none >2 cm without vascular invasion, or a solitary tumor >2 cm without vascular invasion
T3	Solitary tumor >2 cm with vascular invasion, or multiple tumors in one lobe, none >2 cm, with vascular invasion, or multiple tumors in one lobe, any >2 cm, with or without vascular invasion
T4	Multiple tumors in more than one lobe or tumors involving a major branch of portal or hepatic vein(s)
Regional lymph node	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Regional lymph node metastasis
Distant metastasis	
MX	Presence of distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis
Stage grouping	
I	T1 N0 M0
II	T2 N0 M0
III	T1 N1 M0
	T2 N1 M0
	T3 any N M0
IVA	T4 any N M0
IVB	T4 any N M1

Interventional Radiological Treatment Of Hepatic Tumors

I- Local Ablative Therapy

A) Chemical ablation

1) Ethanol injection
2) Acetic acid injection

B) Thermal ablation

1) Radiofrequency ablation
2) Microwave ablation
3) Laser ablation
4) Cryoablation
5) High intensity focused ultrasound (HIFU)

Indicastions for local ablative therapy are non surgical patients

- **Small HCC (<5 cm)**
- **3 nodules each is smaller than 3 cm**
- **NO evidence of vascular invasion**
- **NO extrahepatic spread**
- **Child-Pugh class A or B .**

II- Image –Guided Transcatheter Tumor Therapy

1) **Chemoembolization:**
    - **C-TACE: conventional TACE using lipidol**
    - **DEB-TACE: TACE using drug eluting beads**
2) **Hepatic artery chemoperfusion**
3) **Embolization**
4) **Radioembolization**
    - Patient with main portal vein thrombosis can be safely treated with 90Y radioembolization.

Indications:

- Large/multifocal HCC
- NO vascular invasion
- NO extrahepatic spread
- Child A or B
- Performance status 0-2

❖ Evaluation of Liver Tumor Response After Treatment

Tumor enhancement is used as a biomarker of disease response, as proposed by:

- **American Association for the Study of Liver Disease (AASLD)**
- **European Association for the Study of the Liver (EASL).**

Evaluation of Hepatocellular Carcinoma Overall Response According to EASL Criteria, Modified RECIST, and RECICL

Response Category	EASL Criteria*	Modified RECIST†	RECICL‡
Complete response	Disappearance of enhancing tissue in target lesion(s)	Disappearance of intratumoral arterial enhancement and pathologic lymph nodes	Complete tumor necrosis or complete tumor disappearance
Partial response	≥50% decrease in sum of arterial enhancing area	Decrease ≥ 30% in sum of diameters of viable tissue, taking as reference the baseline sum of the diameters	Tumor necrosis or tumor area reduction between 50% and <100%
Stable disease	Neither partial response nor progressive disease	Neither partial response nor progressive disease	Neither partial response nor progressive disease
Progressive disease	≥25% increase in sum of arterial enhancing area or appearance of new lesion(s)	Increase ≥ 20% in sum of diameters of viable tissue, recorded since treatment started	Tumor area growth > 25% regardless of tumor necrosis, or appearance of new lesion

❖Modified RECIST

Target lesion criteria:

- Measurable lesion >1 cm
- Suitable for repeat measurement
- Shows intratumoral enhancement on contrast-enhanced CT or MRI

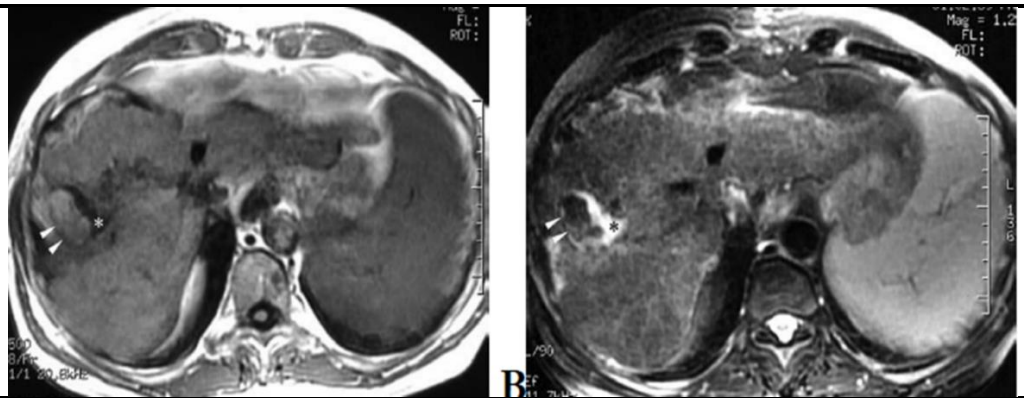
Table 1 Response Assessment by Modified RECIST (adapted from Lencioni et al[8]).

	Terminology	Description
Target lesions	**Complete response (CR)**	Disappearance of any intratumoral arterial enhancement in all target lesions
	Partial response (PR)	At least a 30% decrease in the sum of the diameters of viable (enhancement in the arterial phase) target lesions, taking as reference the baseline sum of the diameters of target lesions
	Stable disease (SD)	Any cases that do not qualify for either PR or PD
	Progressive disease (PD)	An increase of at least 20% in the sum of the diameters of viable (enhancement in the arterial phase) target lesions recorded since treatment started
Non-target lesions	**Complete response (CR)**	Disappearance of any intratumoral arterial enhancement in all non-target lesions
	Stable disease (SD) or incomplete response (IR)	Persistence of intratumoral arterial enhancement in one or more non-target lesions
	Progressive disease (PD)	Appearance of one or more new lesions and/or unequivocal progression of existing non-target lesions
Additional recommendations	**New lesion**	A new lesion can be classified as HCC if its longest diameter is at least 1 cm and the enhancement pattern is typical for HCC. A lesion with atypical radiological pattern can be diagnosed as HCC by evidence of at least 1-cm interval growth.
	Pleural effusion or ascites	Cytopathological confirmation of the neoplastic nature of any effusion that appears or worsens during treatment is required to declare PD
	Lymph nodes in the porta hepatis	Lymph nodes detected at the porta hepatis can be considered malignant if the lymph node short axis is at least 2 cm
	Portal vein thrombosis	Malignant portal vein thrombosis should be considered as a non-measurable lesion and thus included in the non-target lesion group

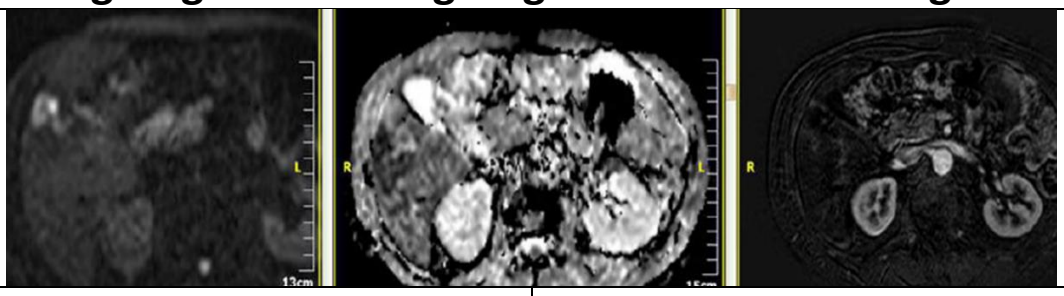
❖Post Hepatic Intervention Imaging Findings:

A- Ablation / Embolized / Targeted Zone

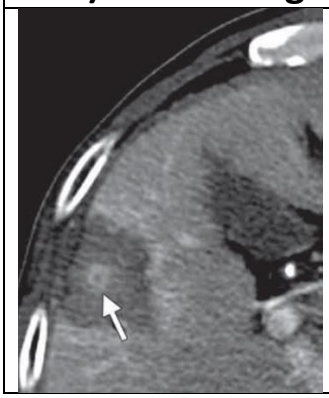
1) Coagulative necrosis	2) Liquefactive Necrosis
CT: Hyperdense	CT: Hypodense
T1: High	T1: Low
T2: Low	T2: High
DWI: Restricted/T2 shine through	DWI: Facilitated
T1+C FS Subtraction: No enhancement	

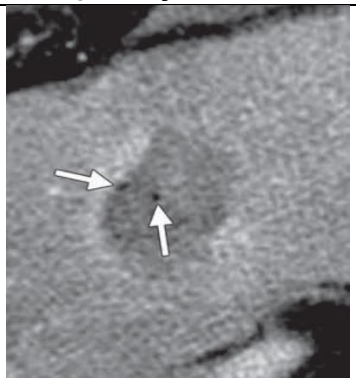


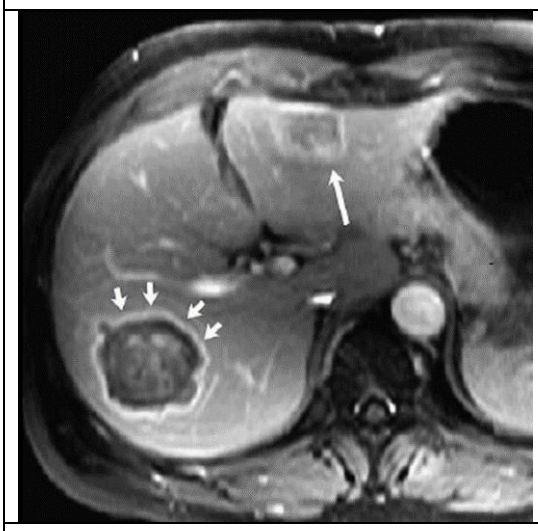
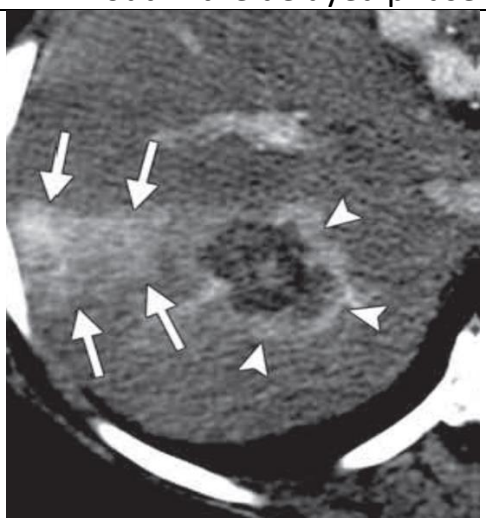
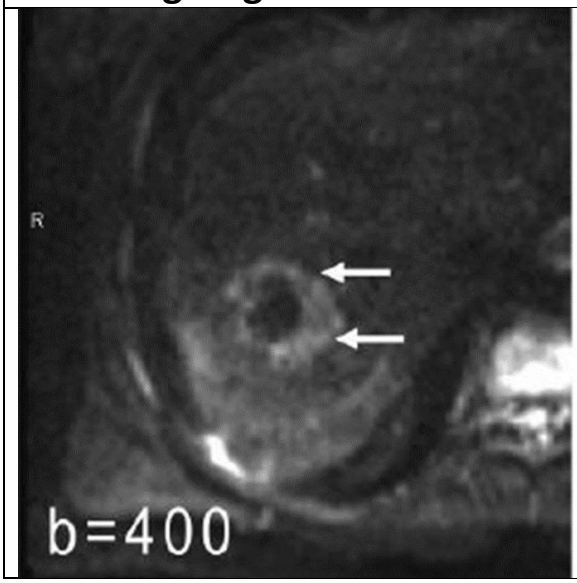
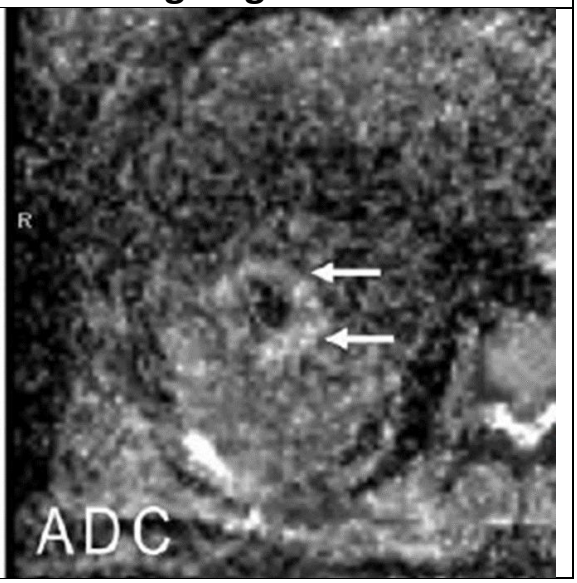
DWI high signal +ADC high signal =T2 shine through



3) Central high-attenuation	4) Tiny air bubbles

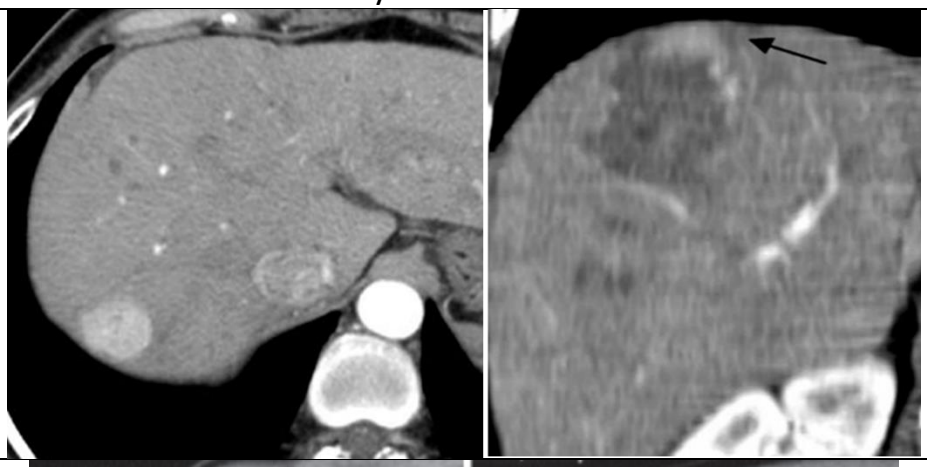


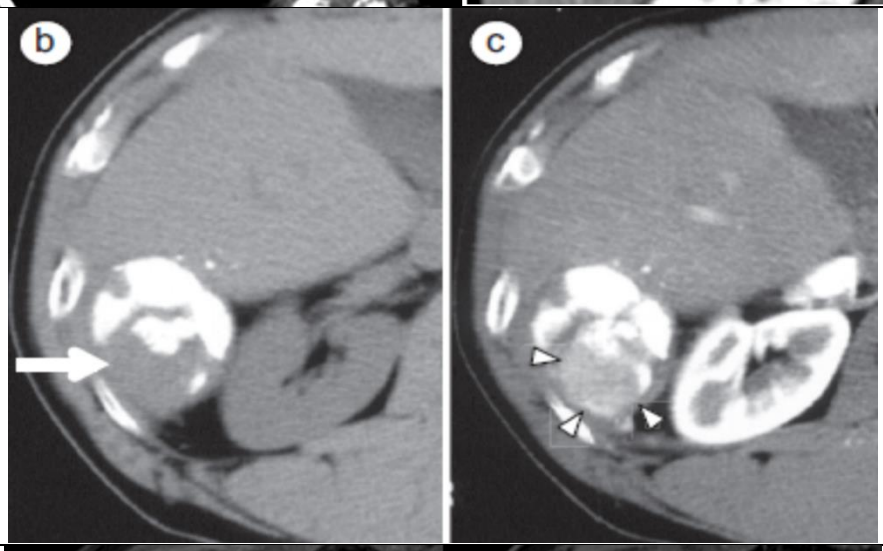


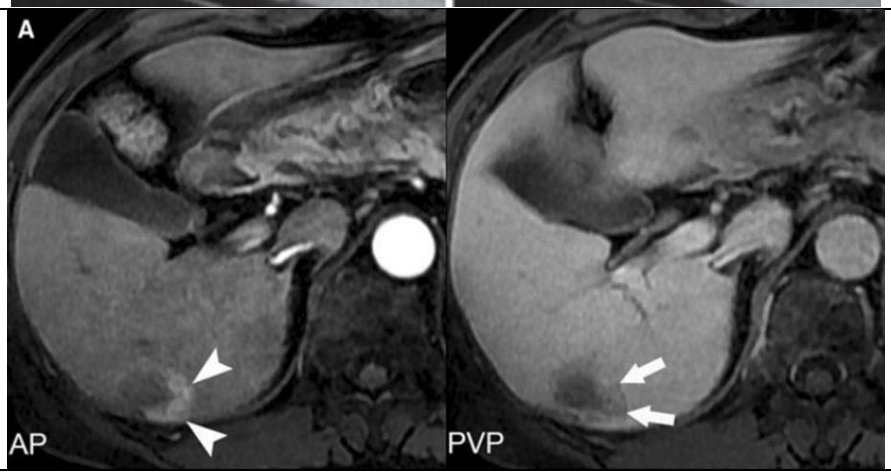
5) Peripheral Rim of Enhancement:	6) Peripheral Wedge-shaped Perfusion
• Thin regular peripheral rim of enhancement	• Transient arterial enhancement with no wash out in the delayed phase.
	
DWI= High signal	**ADC = High signal**
	

B- Residual / Recurrent Tumoral Tissue

- Peripheral thick rim, irregular nodular pathological enhancement
- Arterial enhancement + Delayed washout







C- Complications (See Post trauma Complications)

IV- Hepatic Vascular Disorders

- **Vascular Diseases:**
  - Budd-chiari syndrome
  - Portal vein thrombosis
  - Passive congestion
  - Hepatic infarction
  - HELLP Syndrome
  - Peliosis hepatis

- **Vascular tumors:**
  - Congenital Hemangioma
  - Infantile Hemangioma
  - Infantile hemangioendothelioma
  - Angiosarcoma
- **High Flow:**
  - AVM
  - Arterio-portal Fistula
- **Low Flow:**
  - Congenital Extrahepatic Porto-systemic Shunts (CEPS)
  - Venous / Lymphatic / Venolymphatic malformations
- **Vascular Syndromes:**
  - Hereditary hemorrhagic telangiectasia (osler-weber-rendu syndrome)

- **Hepatic Perfusion Disorders:**
  - Transient Hepatic Attenuation Difference (THAD) / Transient Hepatic Intensity Difference (THID)

1) Budd-Chiari Syndrome

- Budd-Chiari syndrome is defined as lobar or segmental hepatic venous outflow obstruction at the level of:
  - → **Hepatic veins**
  - → **IVC**
  - → **Right atrium.**
- Any age
- More common in women

Primary Causes:

- Congenital causes such as webs and diaphragms,
- Injury
- Infection.

Secondary Causes:

- **Most commonly are thrombotic:**
  - Obstruction of central and sublobular veins after chemotherapy and radiation
  - Obstruction of small centrilobular veins in venoocclusive diseases after bone marrow transplantation
  - Obstruction of the major hepatic veins in patients with hypercoagulability due to oral contraceptive use, pregnancy, polycythemia, or protein C deficiency.
- Nonthrombotic venous obstruction may result from a hepatic or extrahepatic mass.

Pathophysiology:

Hepatic venous outflow obstruction => elevation of sinusoidal pressure + diminished hepatic perfusion => Centrilobular congestion => Hepatic necrosis, fibrosis and cirrhosis.

Clinical Manifestations

i. Fulminant form:

rapid development of hepatic encephalopathy within 8 weeks

ii. Acute form:

rapid onset of jaundice and ascites.

iii. Subacute or chronic form:

more gradual and may be complicated by portal hypertension.

❖ Imaging findings:

A) Morphological Changes:

- Dysmorphic liver
- Caudate lobe hypertrophy (draining vein of caudate is preserved)
- Atrophy of the peripheral segments.
- IVC and hepatic veins thrombosis
- IVC and hepatic veins often are not visible because of collapse or a diminished flow rate.
- Regeneration nodules

B) Parenchymal Changes:

Arterial phase:

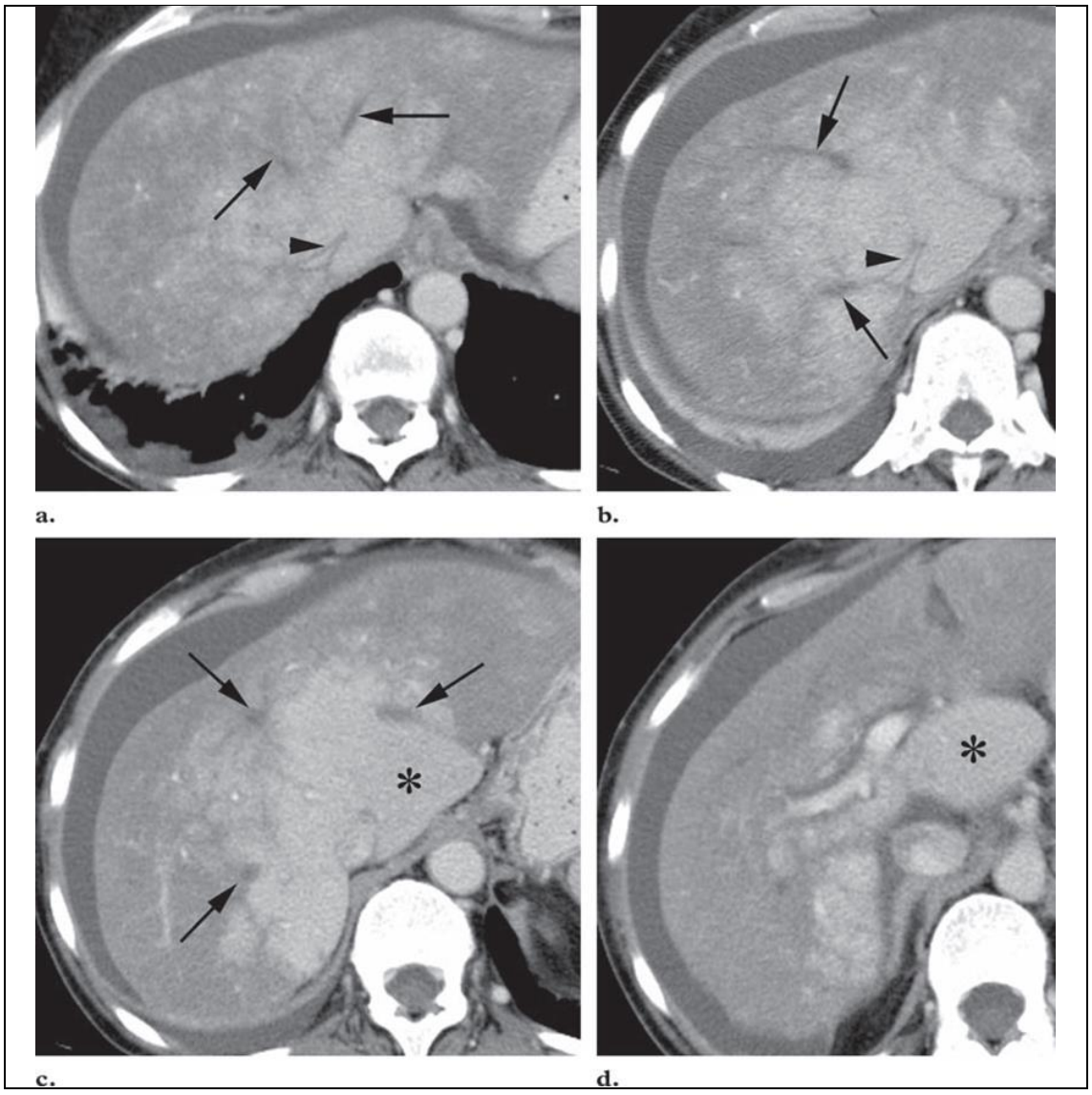
- Early enhancement of the caudate lobe and central portion of the liver around the IVC.
- Decreased peripheral liver enhancement caused by portal and sinusoidal stasis.

Portal/Venous Phase:

- **A "flip-flop" pattern**:
    - Low attenuation of the central part of the liver because of washout
    - Attenuation in the peripheral part of the liver gradually increases with the accumulation of contrast material from capsular veins.
- The hepatic veins and IVC may appear narrowed and hypoattenuating, with hyperattenuating walls.

C) Portal Hypertension Manifestations in chronic stage:

- Splenomegaly
- Portosystemic venous collaterals
- Ascites


a.
b.
c.
d.

2) Portal Venous Thrombosis

It is a major cause of noncirrhotic presinusoidal portal hypertension.

Types:

- **Acute or chronic**
- **Bland or malignant**

Virchow triad:

Hypercoagulability, stasis, and disruption of the vascular endothelium

i) Disruption of the portal venous endothelium:

- Local inflammation/infection (most common)
  - Acute pancreatitis
  - Acute pyelonephritis
  - Ascending cholangitis
  - Chronic inflammatory bowel disease
  - Abdominal surgery, trauma
- Sepsis, Schistosomiasis
- Complication after direct catheterization

ii) Portal venous stasis due to:

- Parenchymal abnormalities such as cirrhosis
- Hepatobiliary malignancies
  - Hepatocellular carcinoma (HCC)
  - Pancreatic carcinoma
  - Cholangiocarcinoma
- Complication after splenectomy.

iii) Hypercoagulable state

- Inherited Prothrombotic Conditions
  - Protein S Deficiency
  - Protein C Deficiency
  - Factor V Leiden Mutation
  - Antiphospholipid Syndrome
- Myeloproliferative Disorders
- Inflammatory Bowel Disease
- Dehydration
- Oral Contraceptive Pills
- Pregnancy

Imaging findings:

US: Echogenic thrombus

T1& T2:

- Acute thrombus will have high signal (see ageing blood on MRI)

+ C

- Complete or partial non-opacification of part of, or the whole, portal vein and its branches
- Rim enhancement:
  - Flow through dilated vasa vasorum
  - Increased flow around periphery of clot
- Bland thrombus: don't enhance
- Tumour thrombus: enhance
- Transient hepatic intensity differences (THID) may be seen in the arterial/early portal phase if the thrombus occludes only a branch of the portal vein, as the hepatic artery takes on the affected lobe/segment's perfusion

PET/CT:

- Bland thrombus: no or slight FDG-avidity
- Tumour thrombus: moderately to highly FDG-avid
- Suggested optimal cutoff value: max SUV 2.3-3.6

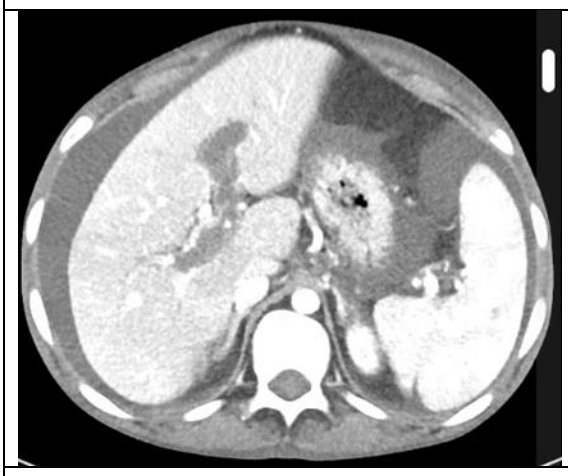
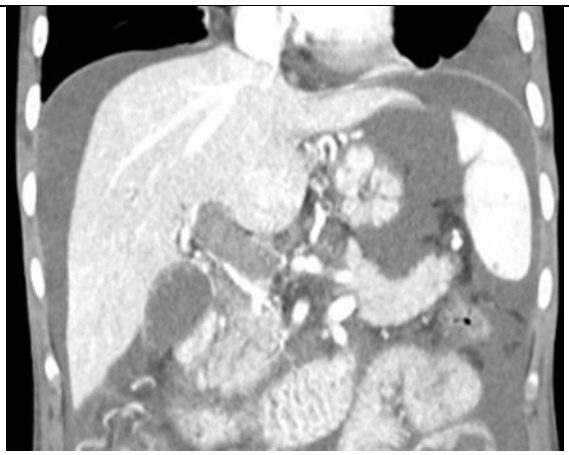
Cavernous transformation

- is the replacement of the normal single channel portal vein with numerous tortuous portoportal venous channels at porta hepatis.
- Enhance during the portal venous phase, and not during the arterial phase (distinguishing it from an arteriovenous malformation)

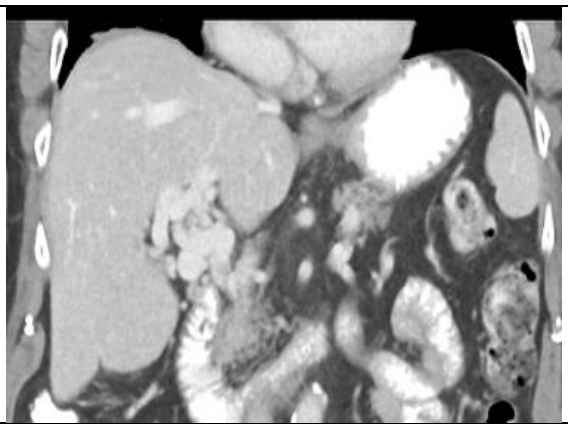
Portal hypertension:

- Portal-systemic collateral pathways
- Splenomegaly
- Ascites

Bland thrombus

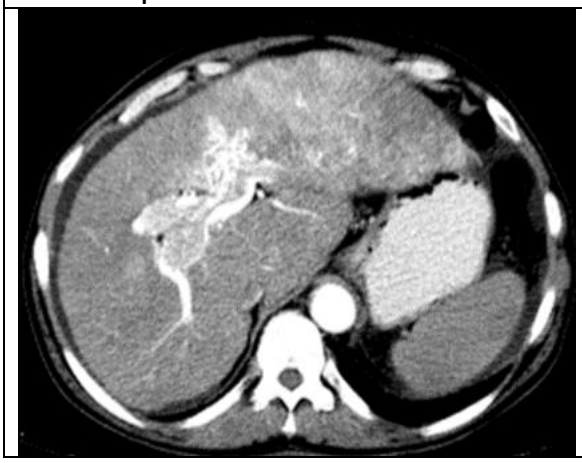



Cavernous Transformation

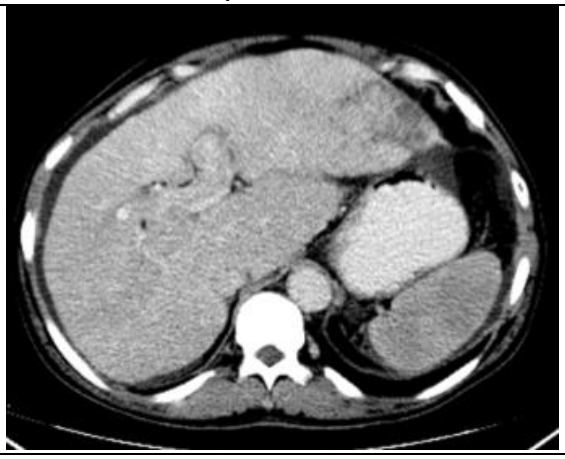


Malignant thrombosis

Arterial phase



Portal venous phase



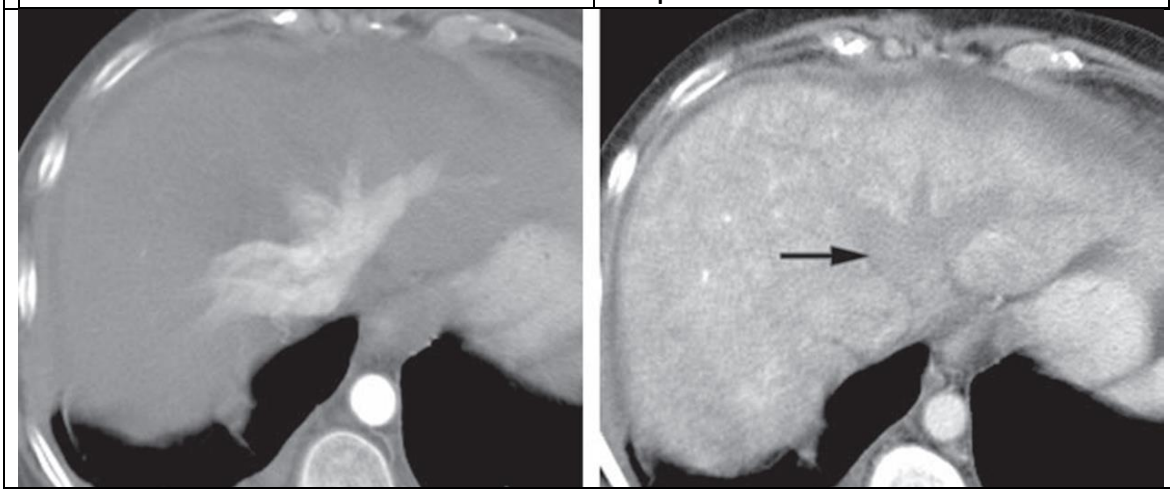
3) Passive Hepatic Congestion

- Passive congestion occurs with the stasis of blood within liver parenchyma as a result of impaired hepatic venous drainage secondary to cardiac disease.

Aetiology:

- Congestive heart failure
- Constrictive pericarditis
- Pericardial effusion
- Cardiomyopathy
- Right-sided valvular disease involving the tricuspid or pulmonary valve.

Arterial phase:	Portal Venous Phase:
• Early enhancement of a dilated IVC and central hepatic veins because of the reflux of contrast material from the right atrium into the IVC.	• Heterogeneous, mottled mosaic pattern of enhancement. • Delayed antegrade enhancement of the hepatic veins (arrow) because of impaired venous drainage due to elevated central venous pressure



4) Hepatic Infarction

- Hepatic infarction is defined as areas of coagulation necrosis from hepatocyte cell death caused by local ischemia, which in turn results from the obstruction of circulation to the affected area, most commonly by a thrombus or embolus.

- Hepatic infarction is uncommon because of the dual blood supply from the hepatic artery and portal vein, as well as extensive collateral vessels

Etiology:

- Iatrogenic (occurring after hepatobiliary surgery, intrahepatic chemoembolization, or a transjugular intrahepatic portosystemic shunt procedure)
- Posttraumatic (occurring after hepatic artery or portal vein laceration).
- Post liver transplantation (3% of adult transplant recipients and 12% of pediatric transplant recipients)
- Secondary to hypercoagulability (in sickle cell disease or antiphospholipid antibody syndrome), vasculitis (in polyarteritis nodosa or systemic lupus erythematosus),
- Infection (in sepsis and shock or rare “emphysematous hepatitis”)

Imaging findings:

- Peripheral wedge shaped areas of low attenuation
- Gas formation has been described in sterile infarcts as well as infected ones.
- Sterile gas is related to the release of intracellular gas from necrotic tissue, an origin similar to that of gas bubbles in hepatic tumors after embolization therapy or radiofrequency ablation

5) HELLP SYNDROME

- HELLP syndrome is a pregnancy-related condition
- It is considered a severe and life threatening form of pre-eclampsia
- It is an abbreviation for:
  - **Haemolysis**
  - **Elevated liver enzymes**
  - **Low platelets**

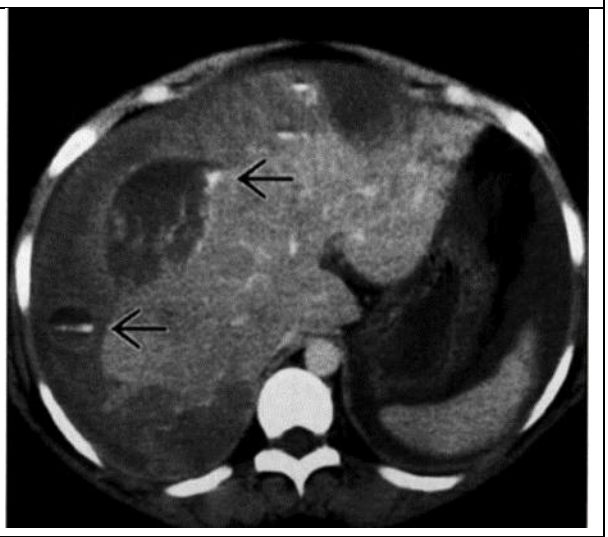
Complications

- Disseminated intravascular coagulation: 20-40% of patients
- Hepatic infarction
- Hepatic hematoma: intra-parenchymal, perihepatic and subcapsular
- Hepatic rupture => Hemoperitoneum
- Placental abruption

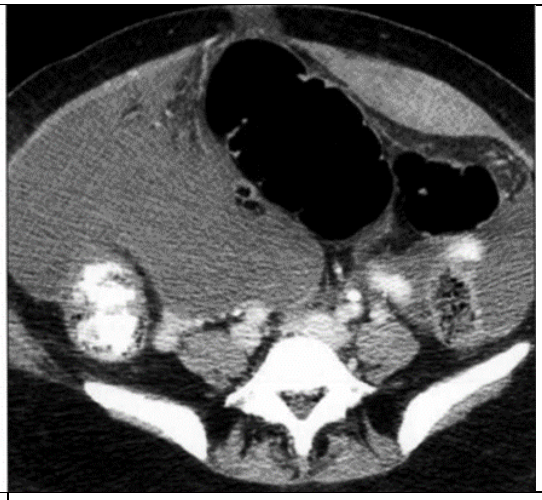
Imaging findings:

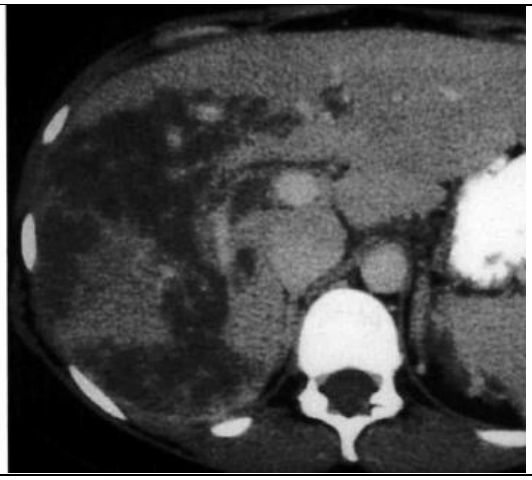
- **Liver hematoma**
  - Well-defined hyperdense or hypodense
  - Nonenhancing
  - Acute: Hyperattenuating (first 24-72 hours)
  - Chronic: Decreased attenuation (after 72 hours)
  - Location: Subcapsular or intraparenchymal
- **Liver infarction**
  - Small or large areas of low attenuation

Subcapsular Hematoma



Hemoperitoneum





6) Peliosis Hepatis

- rare benign disorder that causes sinusoidal dilatation and multiple blood filled lacunae within the liver that vary from 1 mm to several centimeters in diameter

Although the exact origins of this disorder are unknown.

Peliosis hepatis may be secondary to:

- chronic wasting diseases (eg, tuberculosis, leprosy),
- Various malignancies (eg, hepatocellular carcinoma),
- AIDS,
- Drugs (eg, anabolic steroids, corticosteroids, tamoxifen, oral contraceptives, diethylstilbestrol, azathioprine),
- Renal or cardiac transplantation,
- Toxin exposure (eg, polyvinyl chloride, arsenic, thorium oxide).
- Bacillary peliosis hepatis is caused by Bartonella species infection in HIV-positive patients

NCCT:

- The lesions are typically hypoattenuating.

Arterial phase:

- The lesions may appear hypoattenuating in relation to the liver and progressively become hyperattenuating.

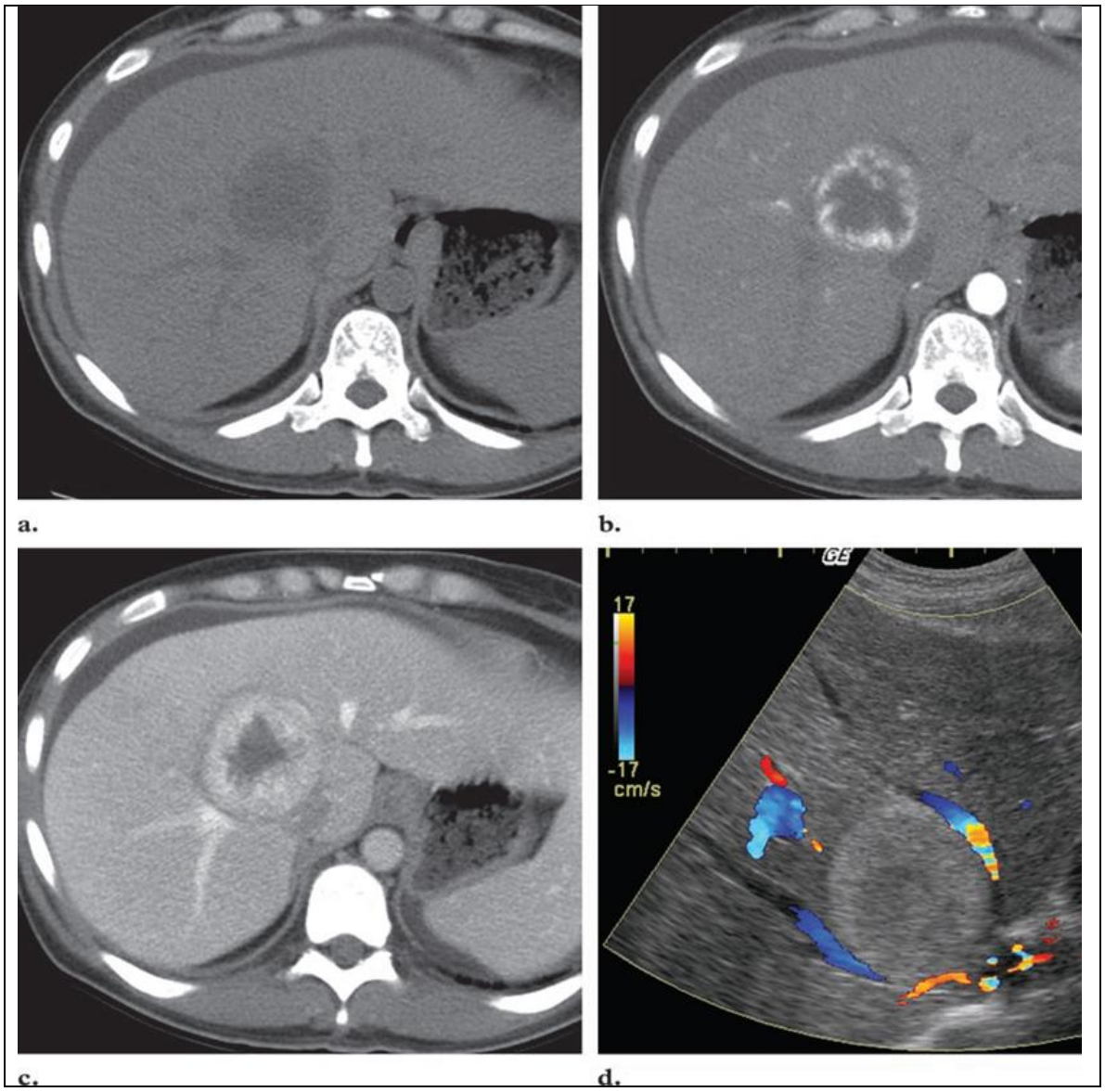
Portal Venous Phase:

- The enhancement may be either complete or early globular vessel-like enhancement with progressive centripetal or centrifugal enhancement without mass effect on adjacent hepatic vessels.

Delayed phase:

- Diffuse increased attenuation may be seen

- ❖ **Thrombosed cavities resemble nonenhancing nodules**
- ❖ **DD: Metastases or Abscesses.**


a.
b.
c.
17
-17
cm/s
d.

7) Hereditary Hemorrhagic Telangiectasia (Osler-Weber-Rendu Syndrome)

- Autosomal dominant multiorgan vascular disorder.
- The condition usually manifests in adults with epistaxis, hemoptysis, or both
- Hepatic involvement is typically diagnosed 10–20 years after the first telangiectasias appear.

Imaging findings:

A. Telangiectasia (63%)
B. Confluent vascular masses (25%)
C. Hepatic Perfusion Abnormalities (THAD)
D. Hepatic Shunts
i. Arteriovenous
ii. Arterioportal
iii. Portovenous

Complications:

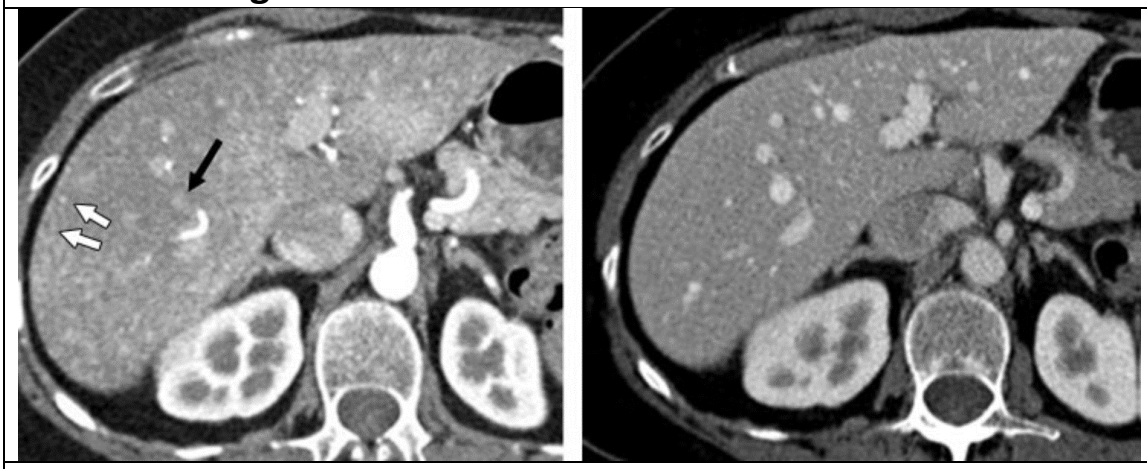
- Hyperdynamic circulation => high-output cardiac failure.
- Extrahepatic shunts, portal hypertension, hepatic portosystemic encephalopathy, biliary ischemia, cholangitis, and liver failure.
- Extrahepatic involvement may manifest with hemoptysis, hemothorax, cerebrovascular accident, or cerebral abscess.
- Most patients experience repeated episodes of epistaxis from nasal mucosal telangiectasias, which bleed spontaneously.

A- Telangiectasia:

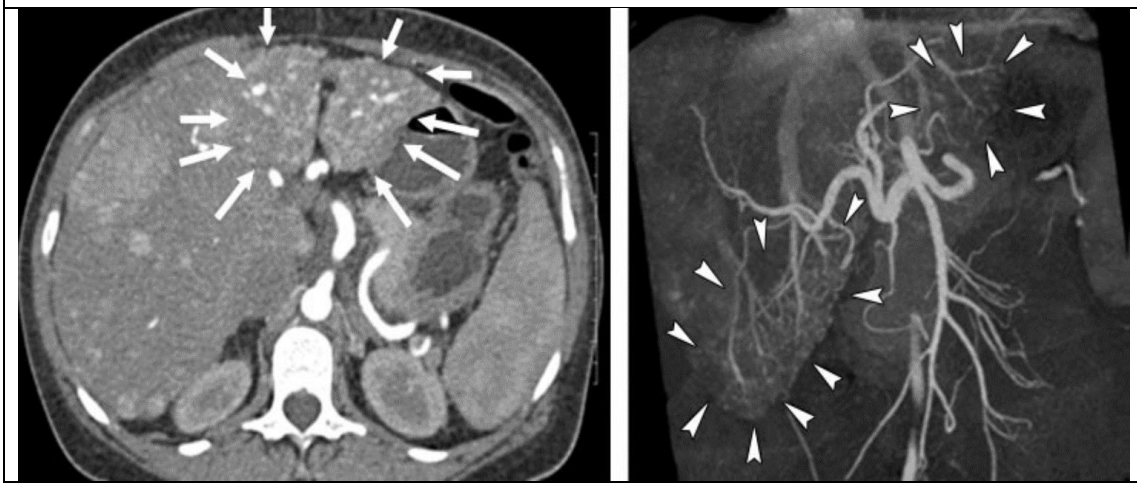
- Telangiectasia is the most commonly seen hepatic lesion
- Focal or diffuse.
- Size: small (few mm - 9 mm)
- Arterial phase: small peripheral subcentimeter hypervascular focus.
- Hepatic phase: becoming isodense with the hepatic parenchyma
- Coronal MIP: helpful in appreciating telangiectases, especially when they exist in proximity to the large vertically oriented vessels, which makes them difficult to appreciate on transverse images

Focal Telangiectasia

Diffuse Telangiectasia

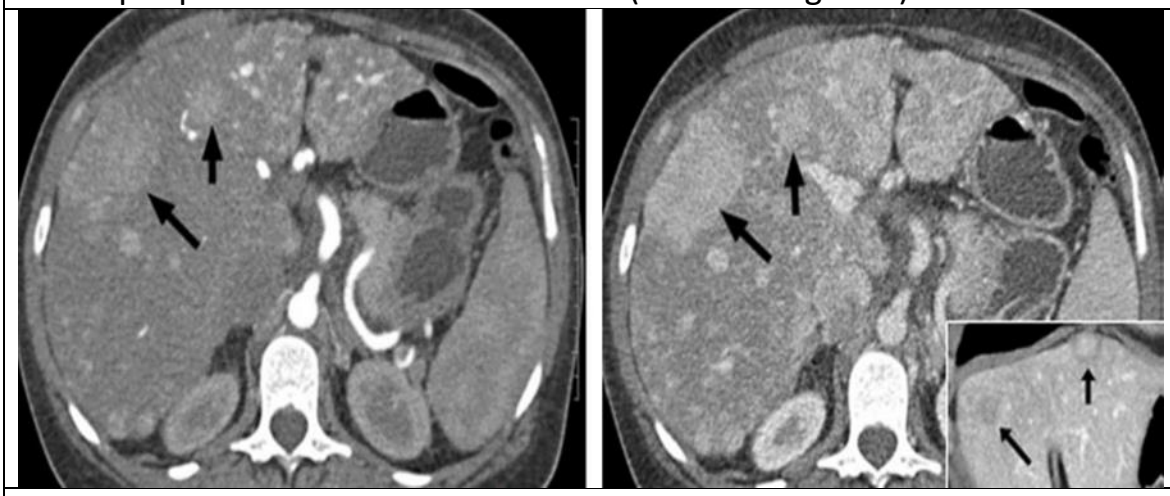


Coronal MIP



B- Large Confluent Vascular Masses

- Large vascular lesion > 10 mm.
- Early enhancement that is seen during both arterial phases, with persistent enhancement in the hepatic phase.
- No peripheral nodular enhancement (DD:Hemangioma)



C- Hepatic Perfusion Abnormalities

- Focal perfusion defects resembling transient hepatic attenuation defects ("THAD" lesions) can be seen alone with peripheral location, triangular shape, and straight margins.
- Indirect sign of other abnormalities, such as an arterioportal shunt, a tiny telangiectasis, or serosal venous drainage

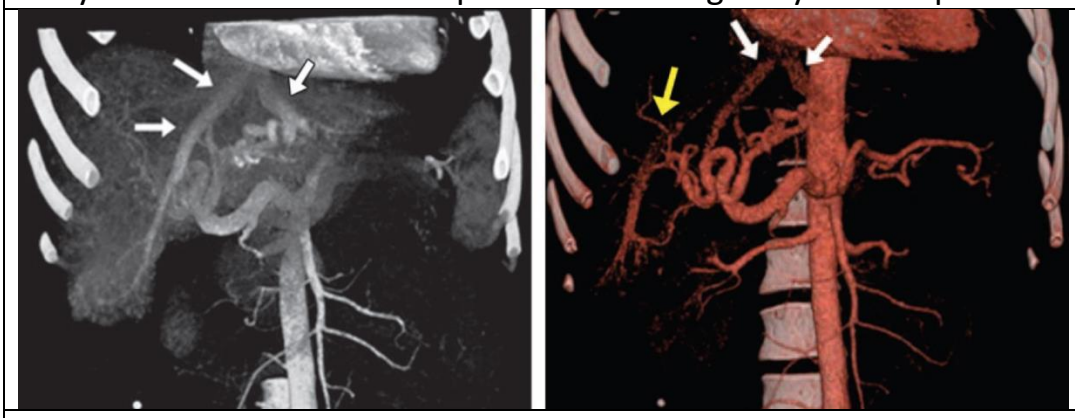
Late arterial phase: show diffuse heterogeneity of attenuation throughout the hepatic parenchyma.

Hepatic phase: disappearance of these abnormalities

D- Hepatic Shunts

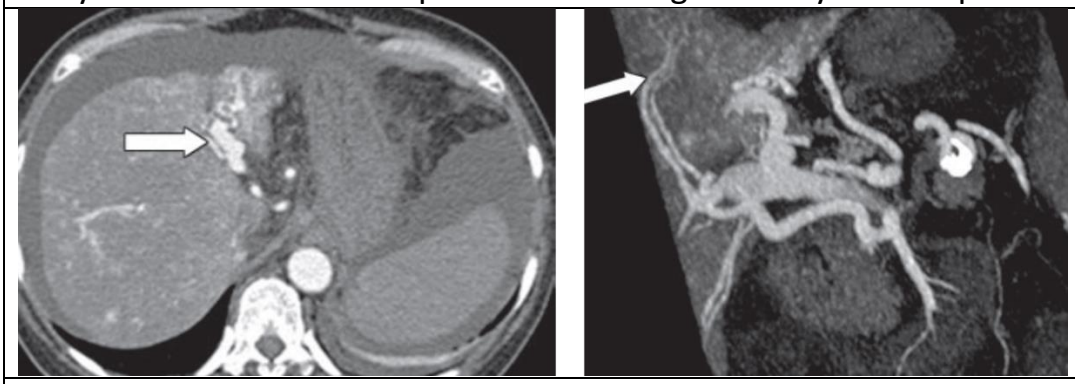
i) Arteriovenous Shunts

Early enhancement of the hepatic veins during early arterial phase



ii) Arterioportal Shunts

Early enhancement of the portal vein during the early arterial phase.



iii) Portovenous Shunts

- are rarely seen in HHT.
- These shunts are seen in the hepatic phase
- Dilated portal vein branch communicating with the large hepatic vein, usually through a focal hepatic mass.
- Portovenous shunts are occult at angiography.

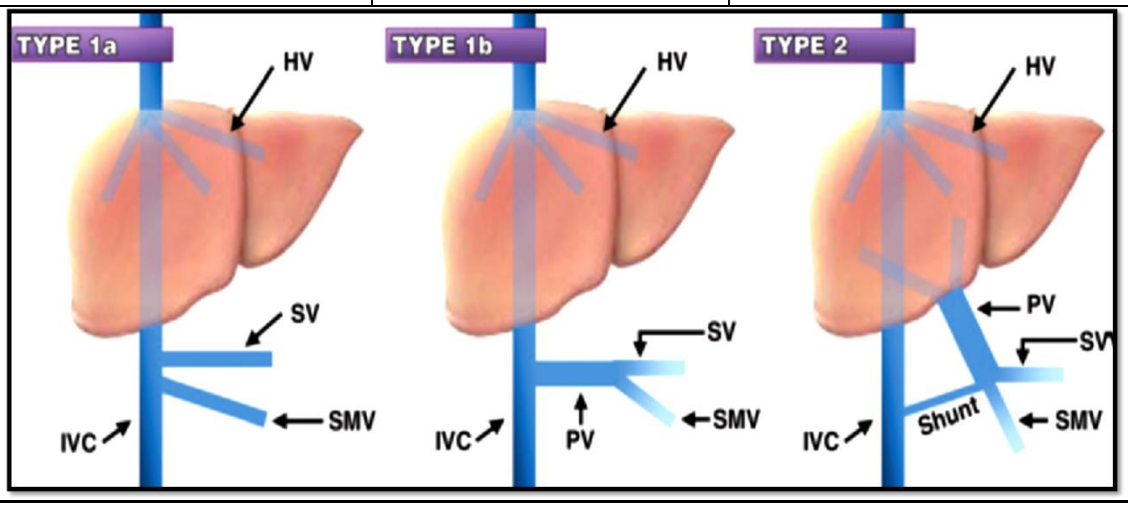
8) Congenital Extrahepatic Portosystemic Shunts

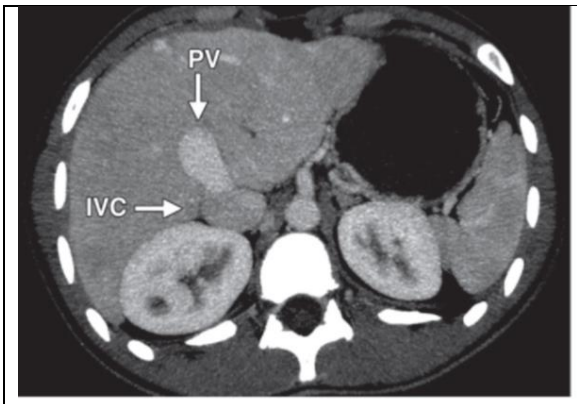
Portomesenteric blood drains into a systemic vein, bypassing the liver through a complete or partial shunt.

Associations:

- Situs ambiguous
- Polysplenia
- Congenital heart defects

Type 1		Type 2
intrahepatic portal venous supply is absent + The portal blood is completely diverted to IVC.		intrahepatic portal venous supply is preserved.
Type 1a	**Type 1b**	
SV and SMV drain separately	SV and SMV drain together after joining to form a common trunk	Partial shunts with some remaining degree of portal perfusion to the liver





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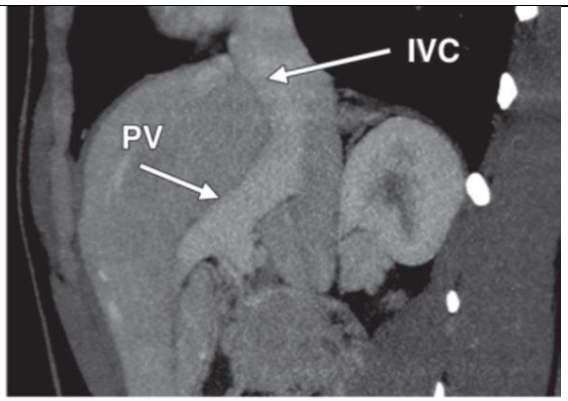

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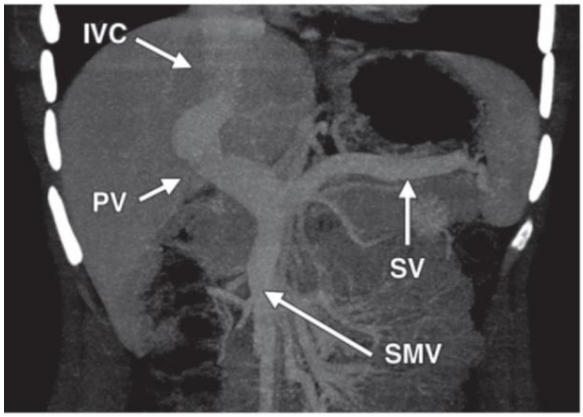

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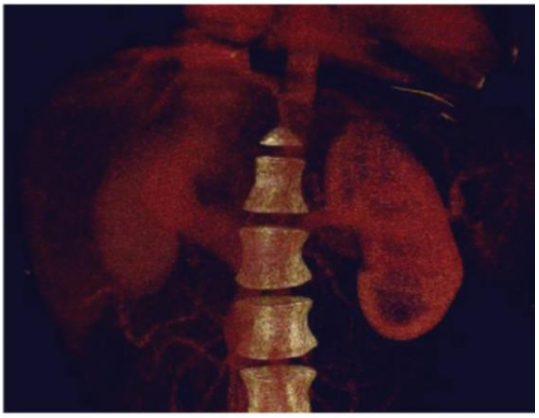

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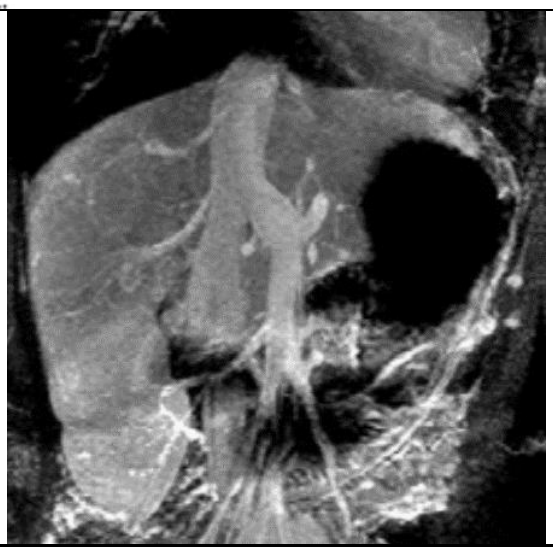
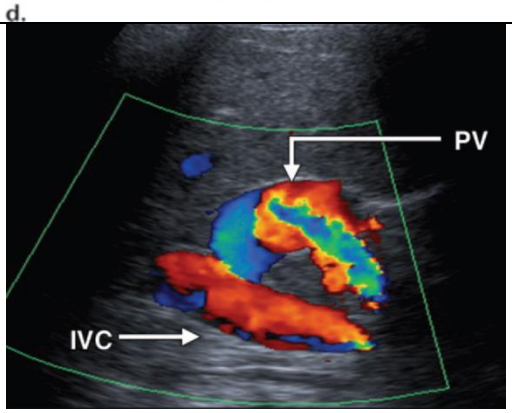




9) Hepatic Perfusion Disorder

Transient Hepatic Attenuation Difference (THAD) / Transient Hepatic Intensity Difference (THID)

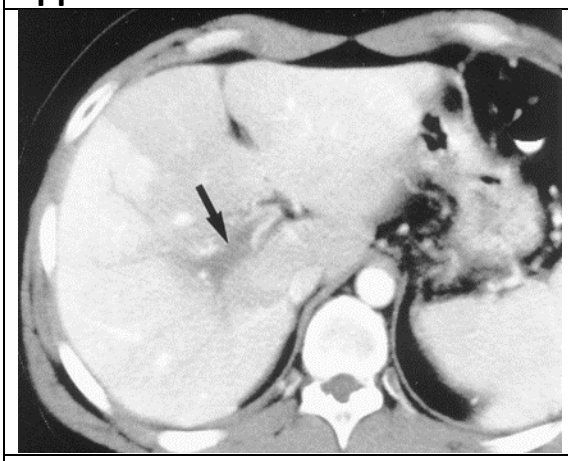
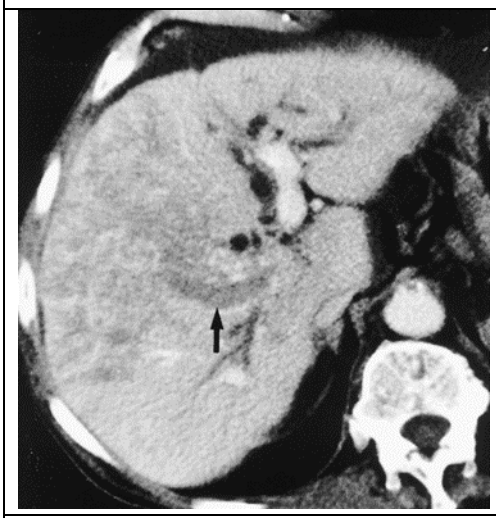
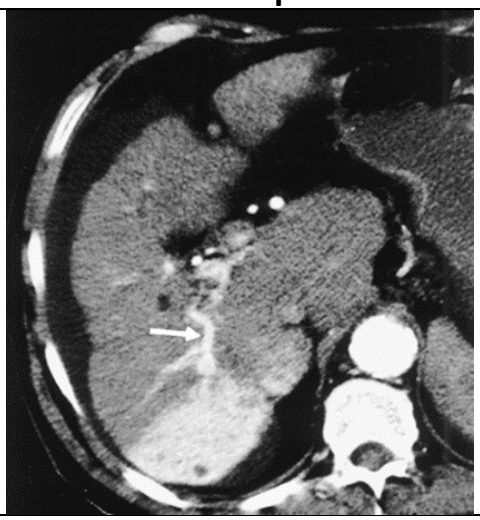
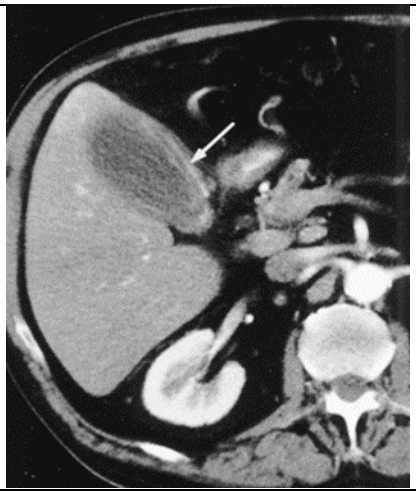
- Reflects a change in the normal dual blood supply of the liver in which the affected site appears as an area of high attenuation on arterial phase and returns to normal on portal venous phase.

Etiology:

- Vascular compromise (portal vein, hepatic vein, hepatic artery obstruction)
- Hepatic vascular disorders: Hereditary hemorrhagic telangiectasia (HHT)
- Liver cirrhosis
- Hypervascular hepatic tumors (steal phenomena)
- Hepatic trauma
- Interventional procedures (Biopsy, RFA, TACE, Embolization)
- Local inflammatory changes (Acute cholecystitis, cholangitis, Abscess)
- Superior vena cava obstruction (Quadrate lobe focal hotspot sign)

Imaging findings:

- Well defined peripheral wedge shaped area
- Straight borders
- Vessel penetration sign: vessel transverse area
- No mass effect
- Triphasic CT => Transient Hepatic Attenuation Difference (THAD):
  - Transient enhancement in the arterial phase
  - Return to normal enhancement in the portal venous phase
- Dynamic MRI => Transient Hepatic Intensity Difference (THID):
  - Transient enhancement in the arterial phase
  - Return to normal enhancement in the portal venous phase

Portal vein obstruction due to appendicitis	**Quadrate lobe focal hot spot sign in SVC obstruction**
	
HCC with PV thrombosis	**HCC with arterio-portal shunting**
	
Post embolization	**Adjacent to cholecystitis**
	

Liver Trauma

- 1st common injured in penetrating trauma
- 2nd common injured in blunt trauma
- CT is currently the diagnostic modality of choice for the evaluation of blunt liver trauma in hemodynamically stable patients.

❖ American Association for the Surgery of Trauma (AAST) CT based Liver Injury Grading System

Grade	Description
I	Hematoma: subcapsular, <10% surface area
	Laceration: capsular tear, <1 cm in parenchymal depth
II	Hematoma: subcapsular, 10%–50% surface area; intraparenchymal, <10 cm in diameter
	Laceration: 1–3 cm in parenchymal depth, <10 cm in length
III	Hematoma: subcapsular, >50% surface area or expanding or ruptured subcapsular hematoma with active bleeding; intraparenchymal, >10 cm or expanding or ruptured
	Laceration: >3 cm in parenchymal depth
IV	Hematoma: ruptured intraparenchymal hematoma with active bleeding
	Laceration: parenchymal disruption involving 25%–75% of a hepatic lobe or one to three Couinaud segments within a single lobe
V	Laceration: parenchymal disruption involving >75% of a hepatic lobe or more than three Couinaud segments within a single lobe
	Vascular: juxtahepatic venous injuries (ie, retrohepatic vena cava or central major hepatic veins)
VI	Vascular: hepatic avulsion

Imaging findings:

1- Initial imaging:-
- **FAST**
- **Plain X-ray**

2- CT

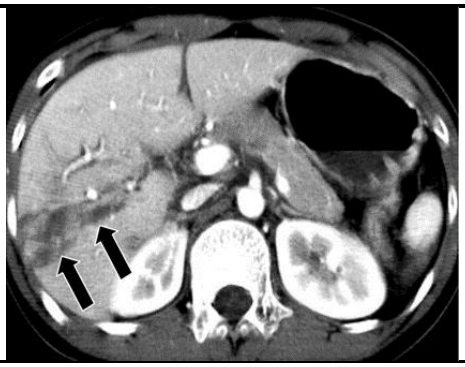
3- Conventional Angiography

4- MRI

5- Isotope Scan

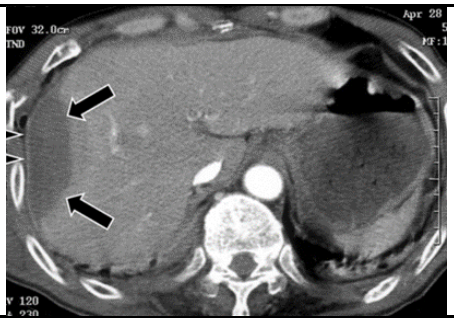
1) Laceration:

- Hepatic laceration is most common type of parenchymal liver injury
- CECT: irregular linear or branching low-attenuation areas
- Superficial (< 3 cm in depth)
- Deep (> 3 cm).



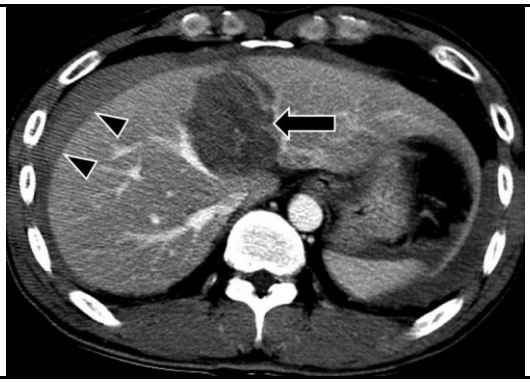
2) Subcapsular Hematoma

- CECT: an elliptic collection of low-attenuation blood between the liver capsule and enhancing liver parenchyma; cause indentation or flattening of the underlying liver margin



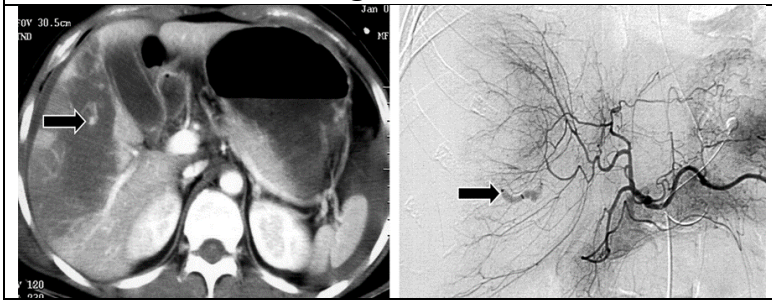
3) Parenchymal hematomas or contusions

- NCCT: Acute hematomas are typically hyperattenuating (40–60 HU) relative to normal liver parenchyma.
- CECT: Focal low-attenuation areas with poorly defined irregular margins in the liver parenchyma.



4) Active Hemorrhage

- Dual phase CECT: Focal high-attenuation areas on initial images that fades into an enlarged, enhanced hematoma on delayed images.



❖ Attenuation Measurements of Hematoma & Vascular Trauma

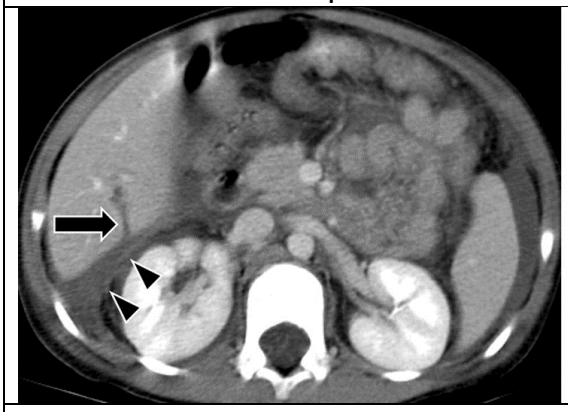
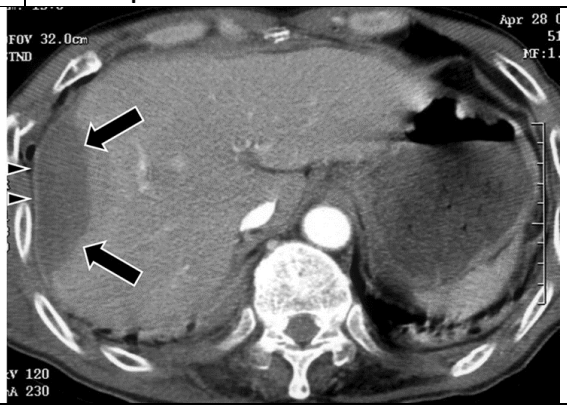
Fluid Type	Attenuation (HU)
Free fluid	0–15
Free blood	20–40
Clotted blood or hematoma	40–70
Active extravasation	Often within 10 HU of adjacent major vessel source

Bleeding Source	Attenuation (HU)
Hematoma with active extravasation	220 ± 5
Nearest artery	230 ± 5
Inferior vena cava	180 ± 5
Portal vein	150 ± 5

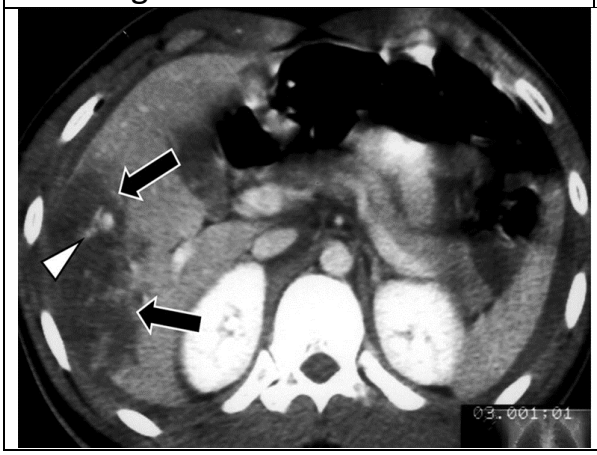
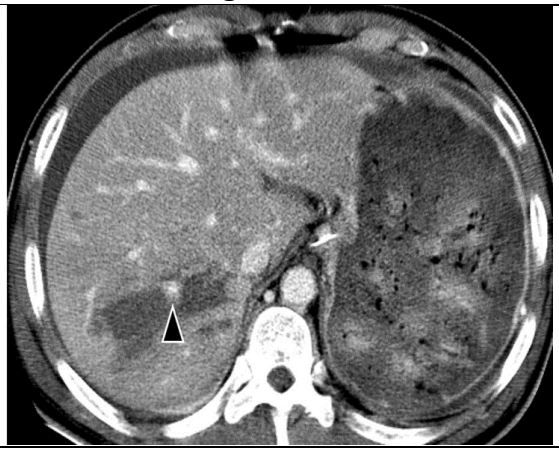
Grade I Hepatic Injury:

Small Capsular tear <1cm depth	Small subcapsular hematoma

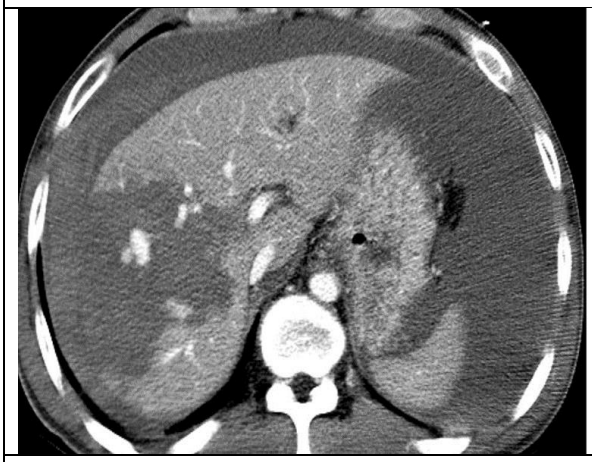
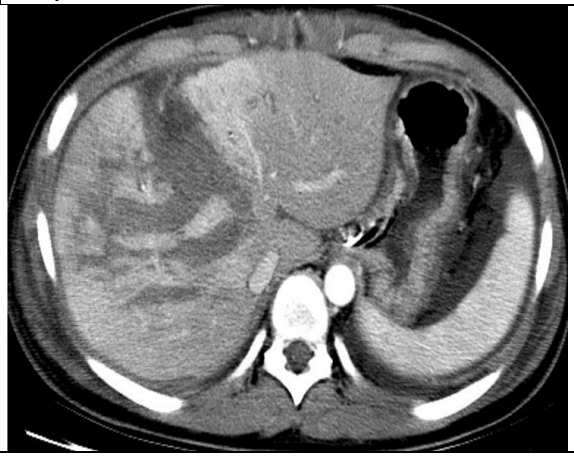
Grade II Hepatic Injury

Laceration 1-3cm depth	Subcapsular hematoma
	

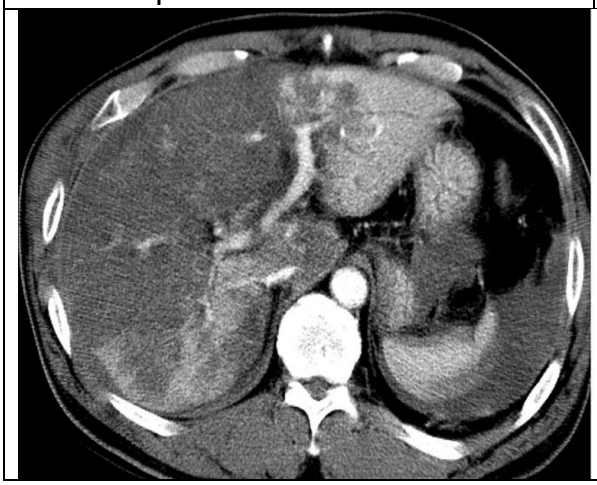
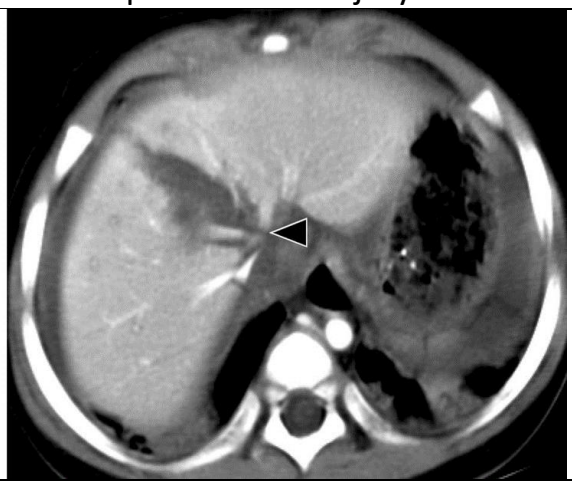
Grade III Hepatic Injury

Subcapsular hematoma with active bleeding	Intraparenchymal hematoma with active bleeding
	

Grade IV Hepatic Injury

Ruptured intraparenchymal hematoma with active bleeding => hemoperitoneum	Multiple hepatic lacerations in the right hepatic lobe => Parenchymal disruption 25-75% of the right hepatic lobe
	

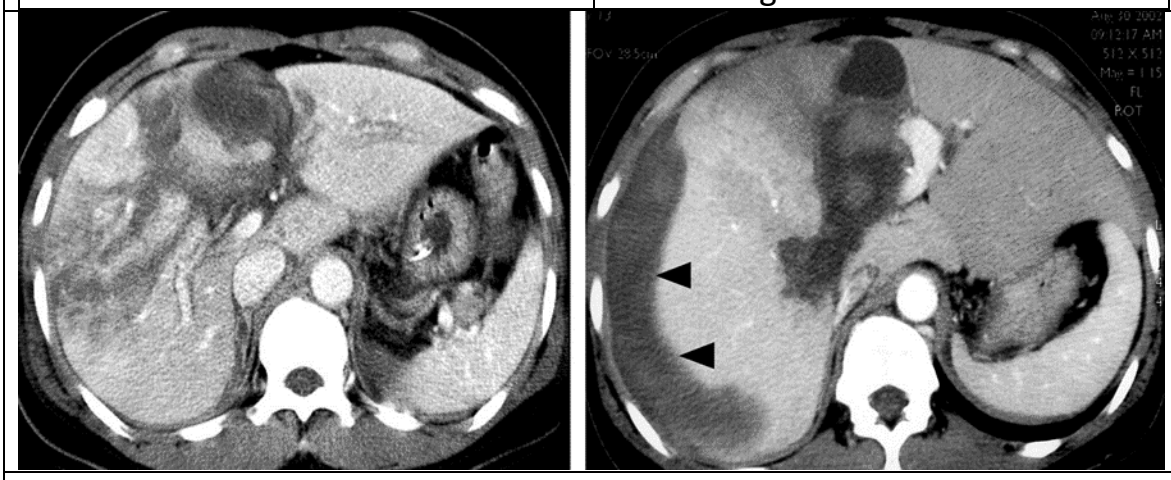
Grade V Hepatic Injury

Large intraprenchymal hematoma => parenchymal disruption of > 75% of the hepatic lobe	deep hepatic laceration that extends into the major hepatic veins = Juxtahepatic venous injury
	

❖Delayed Complications

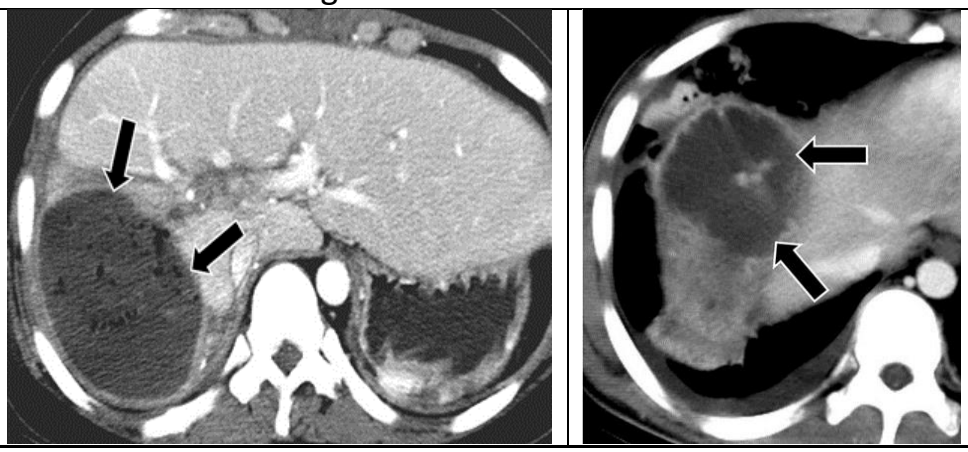
1) Delayed hemorrhage:

Initial contrast-enhanced CT scan shows multiple hepatic lacerations and hematoma in both hepatic lobes	Follow-up shows marked resolution of the parenchymal injury but a newly developed subcapsular hematoma due to delayed hemorrhage



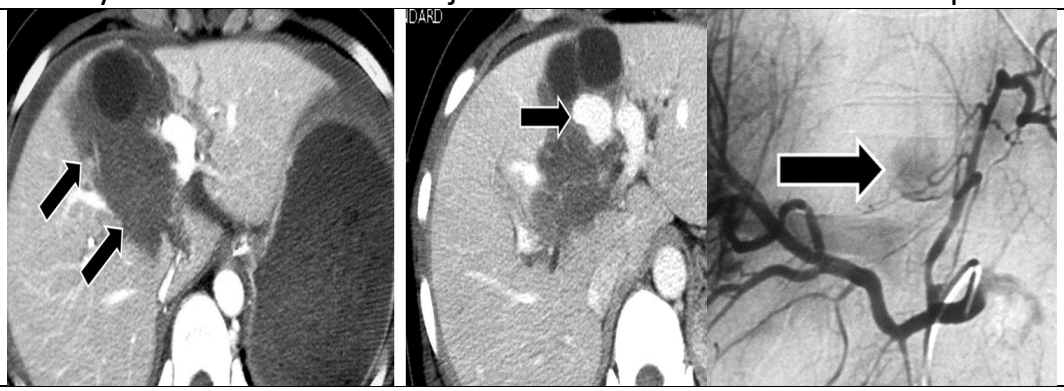
2) Abscess Formation:

Hepatic abscess	**Subphrenic abscess**
A well defined fluid collection + Marginal enhancement + Air loculi within Reactive right side pleural effusion Percutaneous drainage is the treatment	



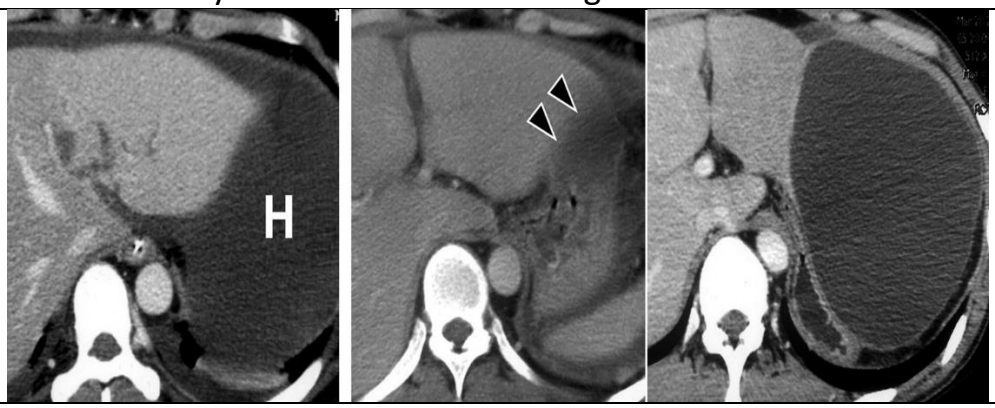
3) Posttraumatic Pseudoaneurysm:

- A well defined rounded or oval shaped sac with neck connects it to the adjacent artery.
- Aneurysm enhancement = adjacent vessel enhancement in all phases



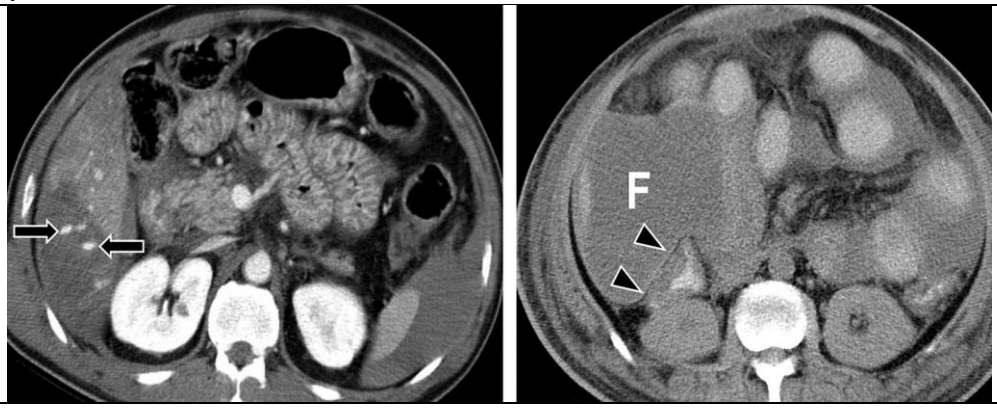
4) Biloma:

- A well defined cystic collection + No marginal enhancement.



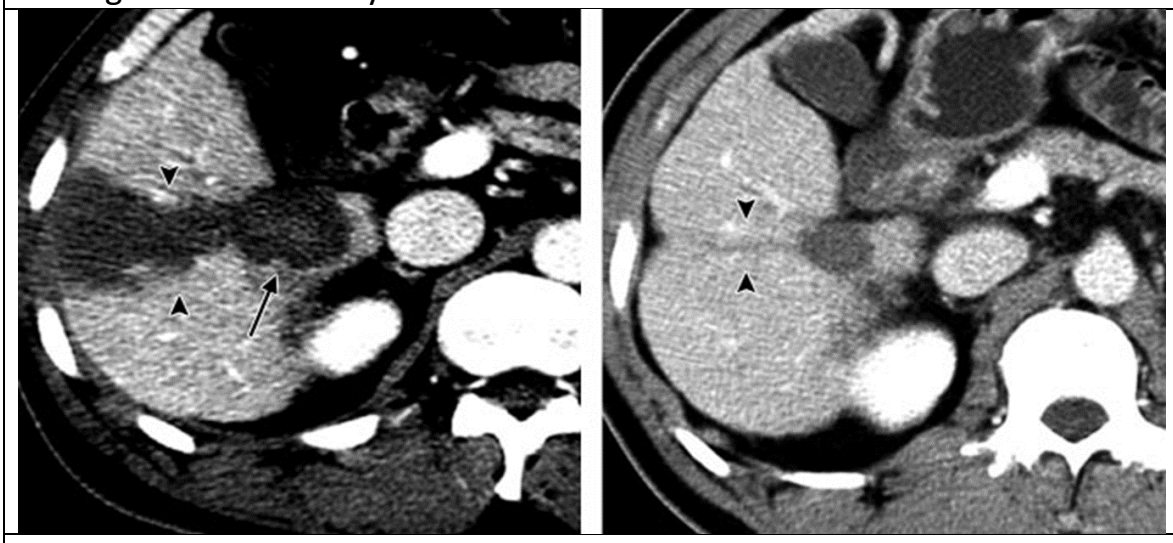
5) Bile Peritonitis:

- Free intraperitoneal fluid
- Aspiration confirmed bile

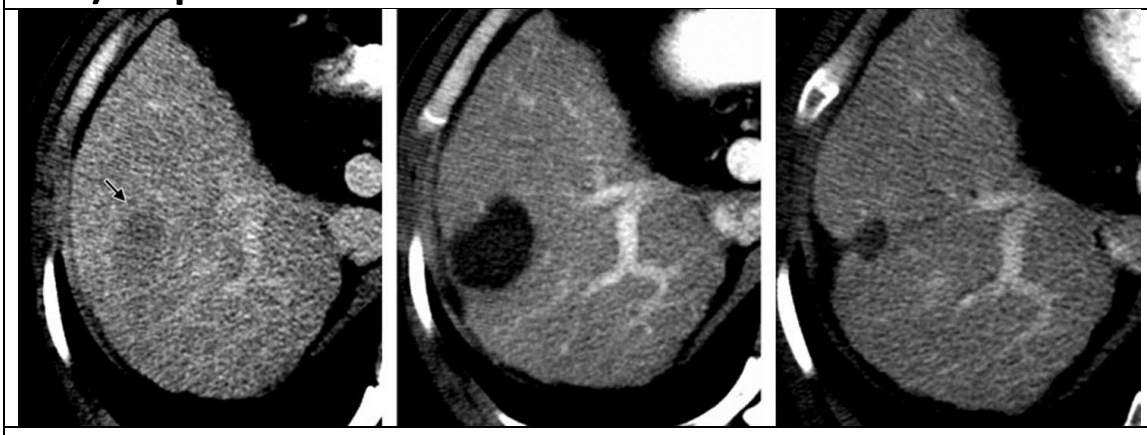


6) Hepatic Infarction:

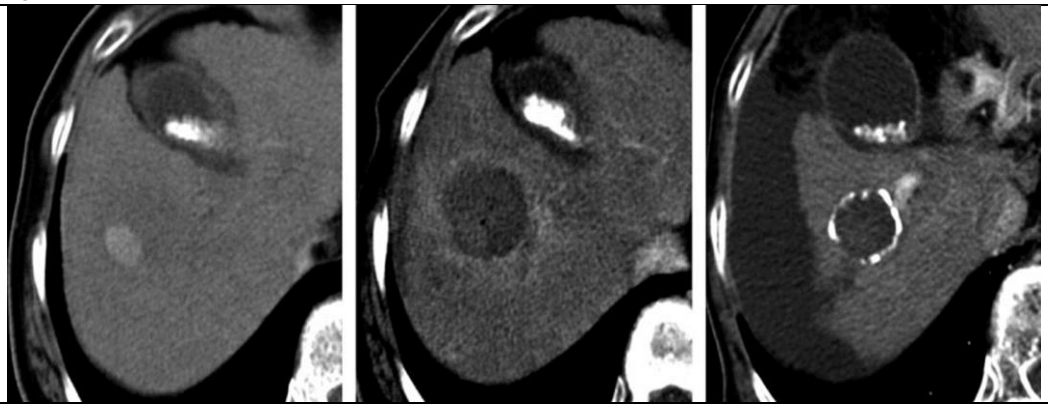
- A wedge shaped hypodense peripheral hypodense area
- No enhancement in all phases
- Regressive course by time



7) Capsular Retraction



8) Calcifications



Gall Bladder

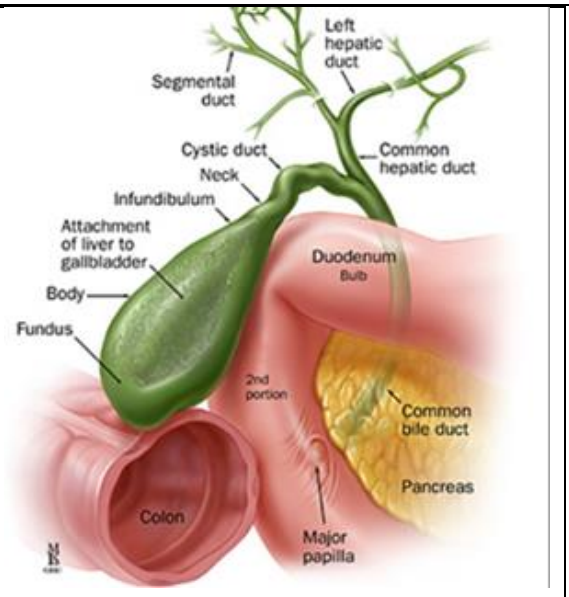
&

Biliary System

Anatomy of Gall Bladder and Biliary Tree

A- Anatomy of Gall Bladder

- Pear-shaped hollow viscus
- Site: Gall bladder fossa at the under surface of the liver; between the right and left hepatic lobes
- Size: variable
  - Length = 7–10 cm
  - Width = 2.5 cm
  - Capacity = 50 ml
  - Wall thickness = 2-3 mm
- Shape: Variable
- Parts: Fundus – Body – Infundibulum (Hartmann pouch) – Neck – Cystic duct



US appearance of GB:

- GB content: Anechoic
- GB wall: Uniform echogenic line

MRI appearance of GB:

- GB wall:
  - T1: Intermediate SI
  - T2: Low SI
  - +C: uniform enhancement
- GB Content = Bile:
  - T1: variable SI depending on concentration:
    - Fasting => Concentrated bile => High T1 SI
  - T2: High SI
  - +C: No enhancement

B- Biliary Tree Anatomy

i) Intrahepatic bile ducts

- Right posterior duct (RPD): segments VI and VII; coursing more horizontally
- Right anterior duct (RAD): segments V and VIII; coursing more vertically
- RAD and RPD unite to from the right hepatic duct (RHD)
- Segmental bile ducts from II-to-IV unite to form the left hepatic duct (LHD)
- RHD and LHD unite to form the common hepatic duct (CHD)

ii) Extrahepatic bile ducts

- CHD is joined by the cystic duct (from the gallbladder) to form the common bile duct (CBD).
- CBD (4 segments:):
  - Supra-duodenal segment: at the free border of lesser omentum
  - Retro-duodenal: crossing posterior to 1st part of duodenum
  - Intrapancreatic segment: cross within pancreatic head
  - CBD unite with the main pancreatic duct to form the ampulla of Vater, which drains at the major duodenal papillae on the medial wall of the D2 segment of the duodenum

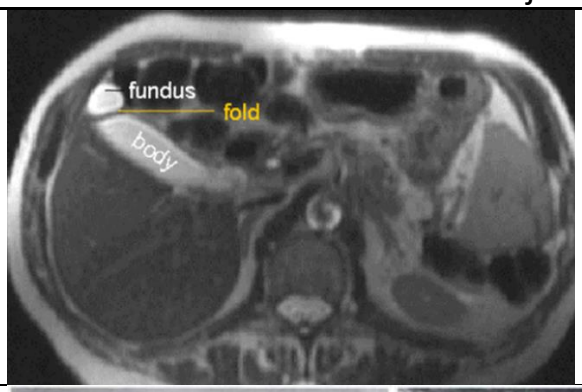
- Normal CBD and CHD diameter < 7 mm
- Post-Cholecystectomy CBD diameter is normal up to 10 mm
- Faintly visible wall at MRI and CT

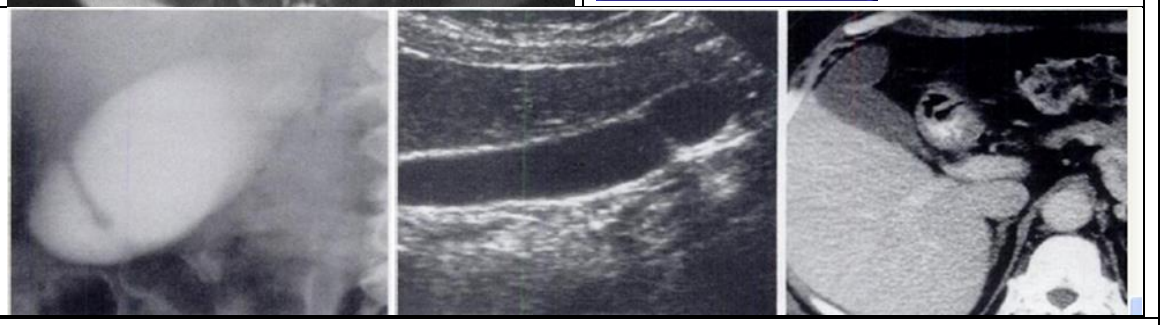
Congenital Anomalies and Normal Variants of Gall Bladder and Biliary Tree

A- Normal Variants of the Gallbladder

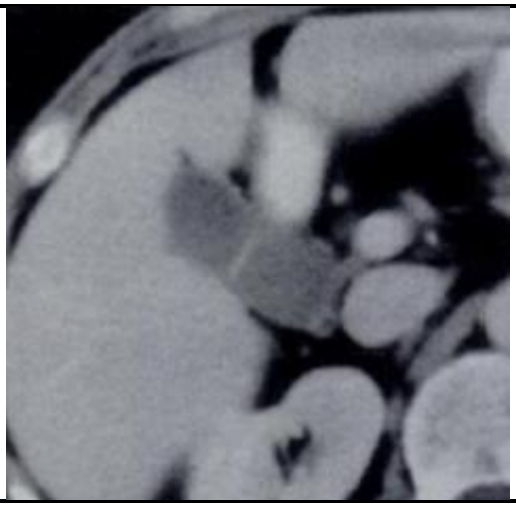
1) Phrygian Cap:

- A fold is seen where the fundus joins the body giving appearance of cap



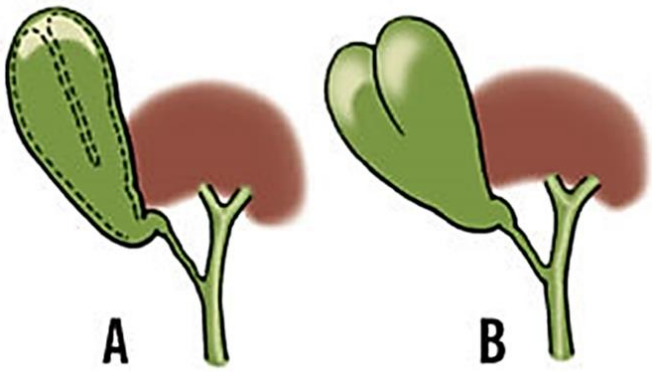


2) Gall Bladder Stations



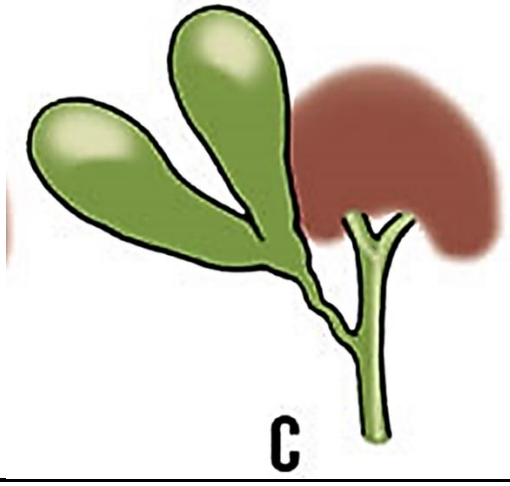
3) Septate Gallbladder

- A longitudinal septum divides the gallbladder into two chambers.
- There may be no external trace of the septum or there may be a fundic cleft extending toward the neck (11.3%)



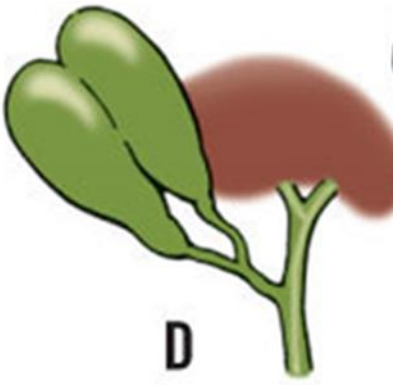
4) Bilobate "V" gallbladder

2 gallbladders, separated at the fundus, are joined at the neck + single, normal cystic duct (8.5%).

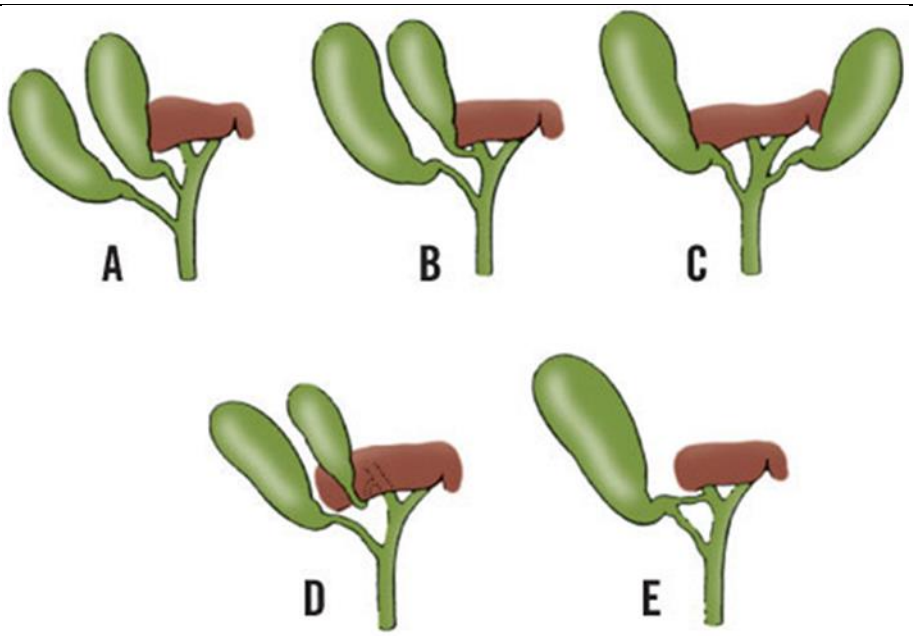


5) "Y" duplication

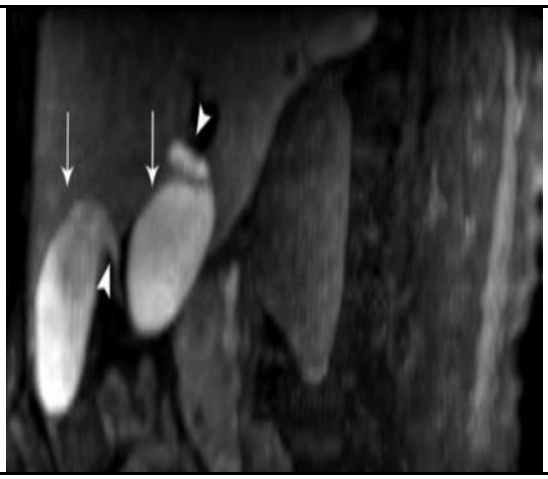
2 separate gallbladders + 2 cystic ducts join to form a common cystic duct before entering the common bile duct.



6) Duplicate Gall Bladder

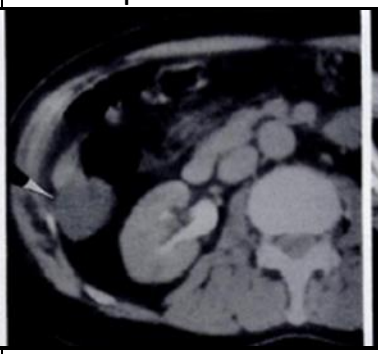
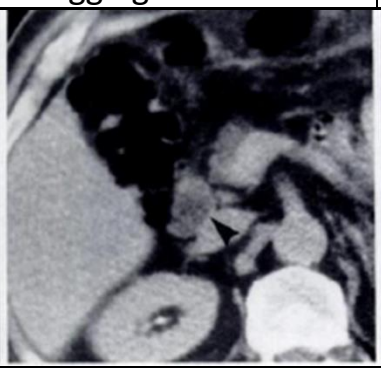
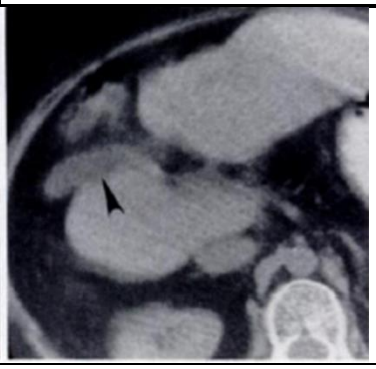


2 separate gall bladders +
2 separate cystic ducts

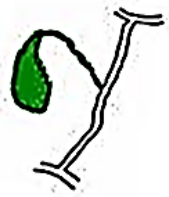


7) Ectopic Gall Bladder

i. Below left lobe of liver, most common
ii. Intrahepatic
iii. Retrohepatic
iv. Within falciform ligament
v. Within inerlobar fissure
vi. Suprahepatic
vii. Anterior abdominal wall
viii. In chest
ix. Not in groin

Intra-hepatic	Sub-hepatic	Pelvic
		
Epigastric	**Hangging**	**Lateral abdominal wall**
		

B- Normal Variants of Cystic Duct



Lateral angular insertion (75%)

Anterior spiral

Posterior spiral

Low insertion (10%)

Parallel with CBD with common sheath (14 - 23%)

Insertion into ampula of vater

High insertion

Insertion into right hepatic duct (0.006 - 0.01%)

Insertion into left hepatic duct

Cystohepatic duct (1 - 2%)

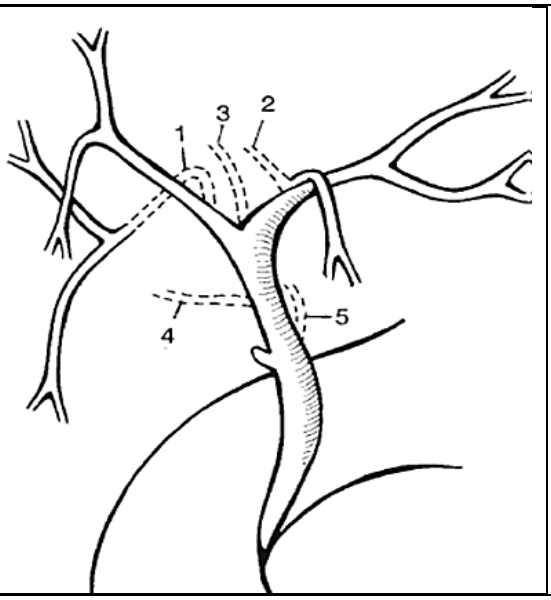
Double cystic duct

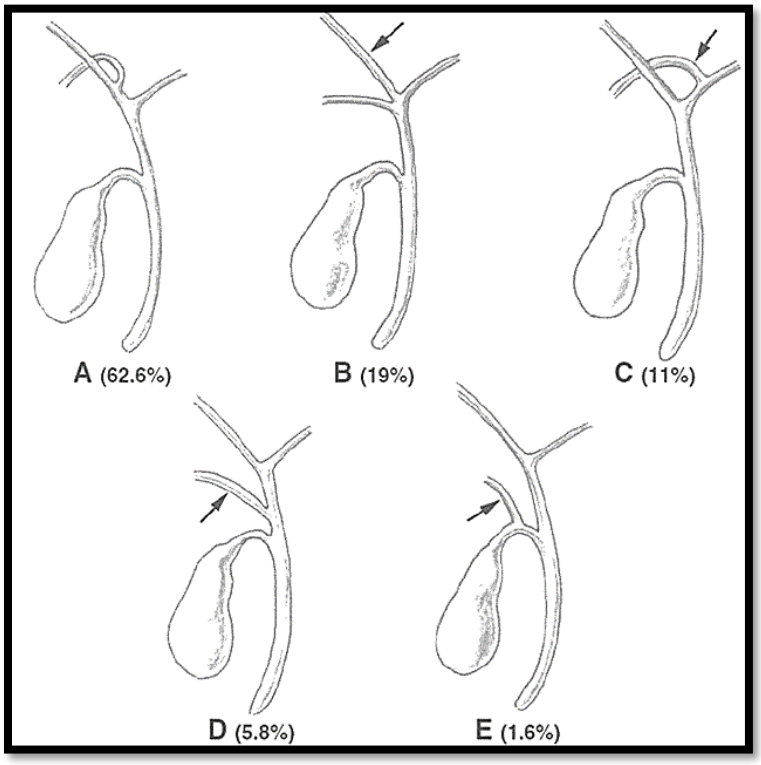
Absent cystic duct

C- Normal variants of Intrahepatic Biliary

Right posterior hepatic duct (RPHD) Drainage into:

1) Right anterior duct
2) Left hepatic duct
3) Trifurcation
4) Lateral side of common hepatic duct (CHD)
5) Medial side of common hepatic duct (CHD).
6) Cystic duct



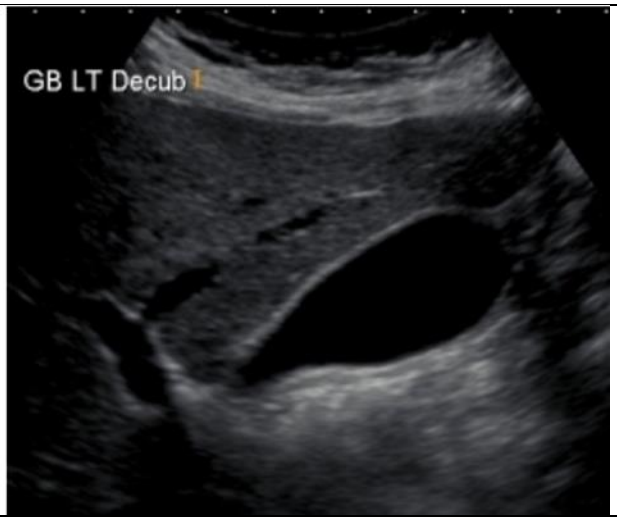


D- Todani system classification of the congenital cystic dilatation of the biliary tree (Disscused Later)

Imaging Techniques of Gall Bladder and Biliary System

❖ US

- Patient preparation: 4hrs fasting
- Patient position: Left lateral decubitus
- Probe scan plane:
  - Subcostal oblique view
  - Intercostal oblique view
- 2 planes
  - GB long axis: sweep from side to side
  - GB Transverse view: from neck to fundus



❖ MRI Protocol

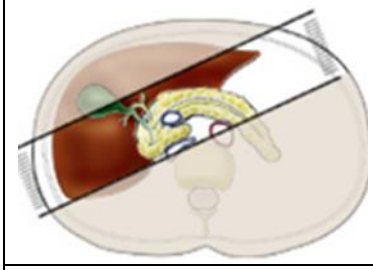
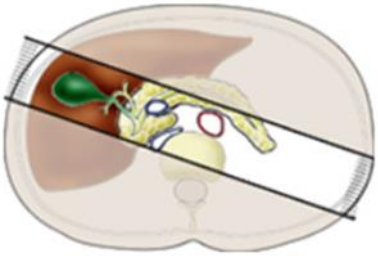
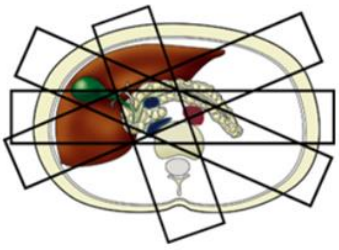
Conventional MRI:

- Axial T1 in-of-phase
- Axial T1 out-of-phase
- Axial T2 SSFSE
- Axial T2 FS
- Coronal T2 SSFSE

Dynamic MRI: (Discussed Before)

MRCP:

- MRCP Radial Thick slab:
  - Oblique radial T2 SSFSE T2 (14 sections)
- MRCP 2D thin slice:
  - Right anterior oblique T2 SSFSE (Slice thickness = 5 mm)
  - Left anterior oblique T2 SSFSE (Slice thickness = 5mm)
- MRCP 3D:
  - Coronal T2 SSFSE (Slice thickness = 1.4mm)

MRCP 2D Thin Slice Right Anterior oblique Rotate 20-30 anticlockwise Centre block on CBD Exclude GB	MRCP 2D Thin slice Left Anterior oblique Rotate 20-30 clockwise Centered on the CBD and include entire GB	MRCP (Thick Slab) 20-30 degree interval
		

Contrast-Enhanced MR Cholangiography

- Hepatobiliary specific contrast agent: Gd-BOPTA or MnDPDP
- Axial and coronal 3D breath-hold fat-suppressed GE T1
- Slice thickness = 2.4 mm
- Delayed phase: 30–90 minutes after contrast material injection

❖ CT Protocol:

Standard CT:

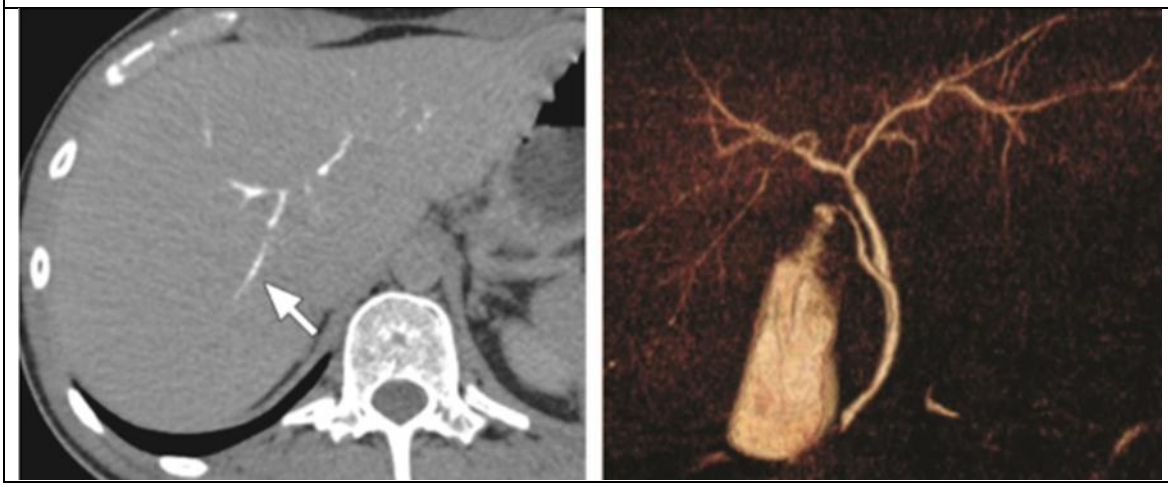
- NCCT
- Portal venous phase (60-80sec)

Triphasic CT:

- NCCT
- Late arterial phase (35sec): Detect suspected pancreatic lesions
- Portal venous phase (60-80sec): detect suspected hepatic lesions
- Delayed phase: 3-5 min

Contrast- enhanced CT Cholangiography:

- CT of the biliary tract can be augmented with use of positive biliary contrast material which can be introduced by:
  - Direct injection into the bile ducts via a percutaneous transhepatic biliary catheter or by means of ERCP.
  - Noninvasive opacification of the biliary tract can be achieved with the administration of intravenous contrast materials that are excreted into the bile.
    - 25 mg of intravenous diphenhydramine
    - Followed by a 30-minute intravenous drip infusion of 20 mL of 52% iodipamide meglumine diluted in 80–100 mL of normal saline solution
    - Scan the liver 15 minutes after completion of contrast material infusion



Gallbladder Wall Thickening

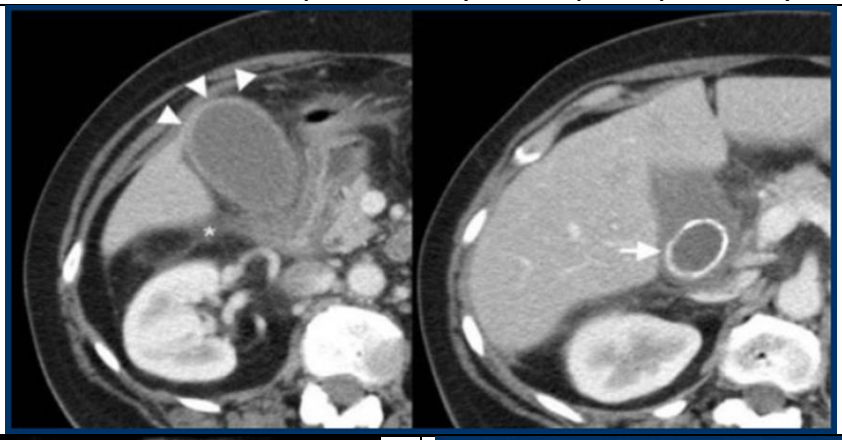
Primary GB disease	Secondary GB involvement
• **Acute cholecystitis** • **Acalculous cholecystitis** • **Chronic cholecystitis** • **Xanthogranulomatous cholecystitis** • **Porcelain gallbladder** • **Adenomyomatosis** • **Gallbladder carcinoma**	• **Liver cirrhosis** • **Hepatitis** • **Pancreatitis** • **Congestive right heart failure** • **Renal failure**

A- Acute Calcular Cholecystitis

The clinical presentation is characterized by right upper quadrant tenderness, abdominal pain, fever, and leukocytosis.

Imaging findings:

- Dilated gall bladder
- Impacted gallstone in gall bladder neck or cystic duct
- Positive sonographic Murphy sign
- Gallbladder wall thickening > 3mm
- Pericholecystic fluid collections
- Edema of the surrounding liver tissue may be found
- Stranding of the surrounding fat
- +C: Increased enhancement of the gallbladder wall, adjacent fat, and surrounding liver Parenchyma
- Transient enhancement of pericholecystic hepatic parenchyma

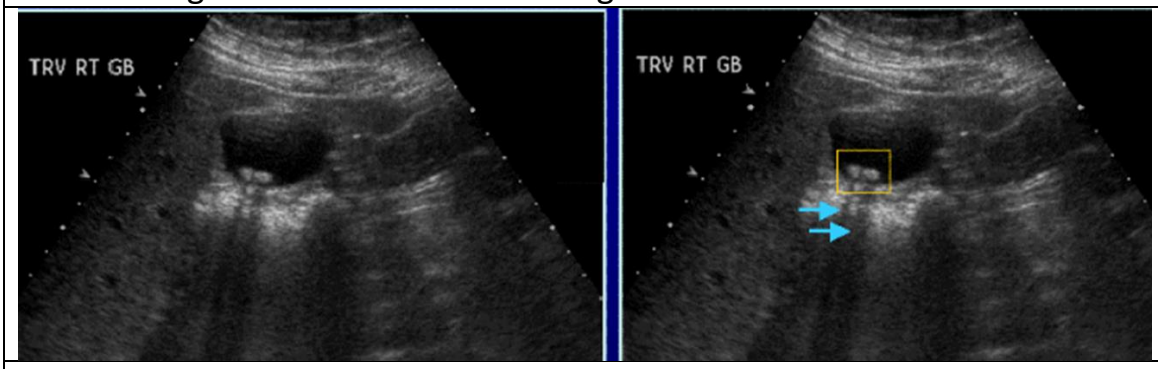


❖Gall Bladder Stones

- Female – Forty – Fatty – Fertile – Flatulent

US:

- Most commonly used modality in the evaluation of gallstone disease.
- High specificity (>95%)
- High sensitivity (95%)
- Echogenic + Posterior shadowing

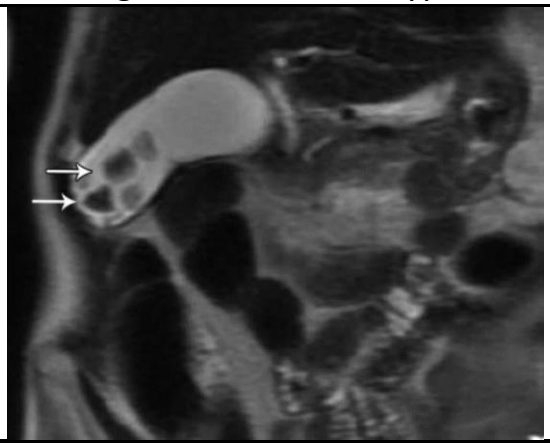


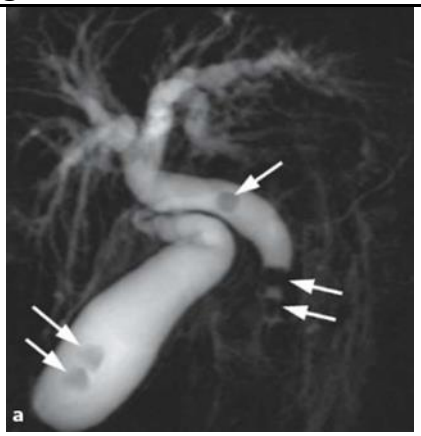
CT:

- Not modality of choice
- Attenuation of calculi varies from less than water density, through soft tissue, to dense calcification

MRI:

- Cholesterol stone: T1 hypo + T2 Signal void
- Pigment stones: T1 hyper + T2 signal void

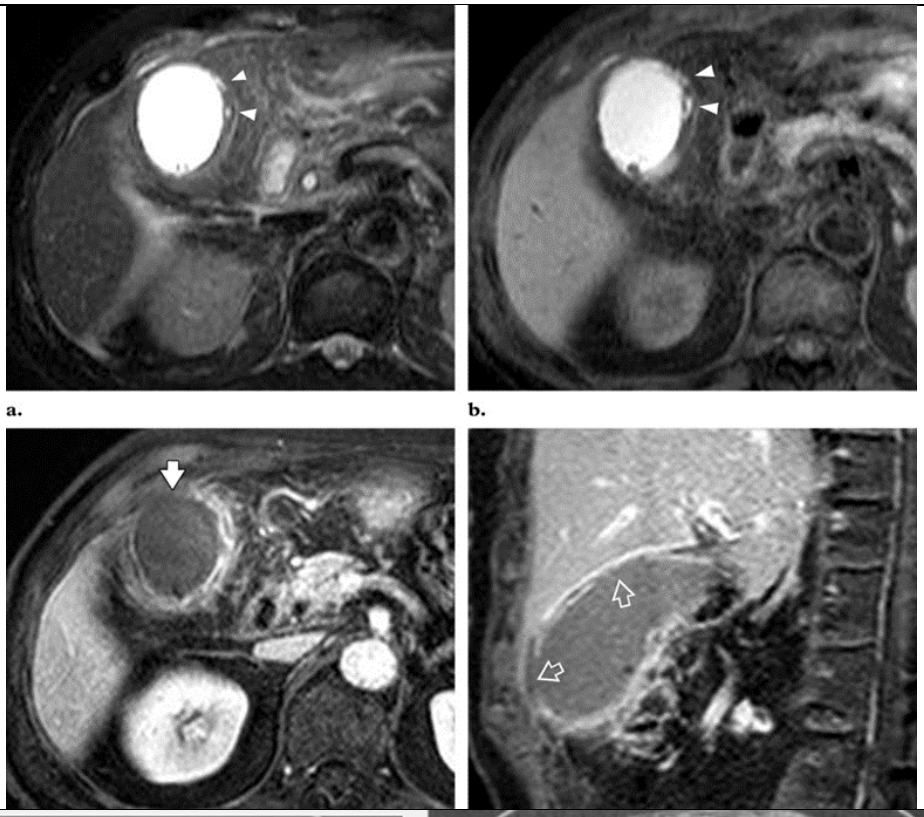


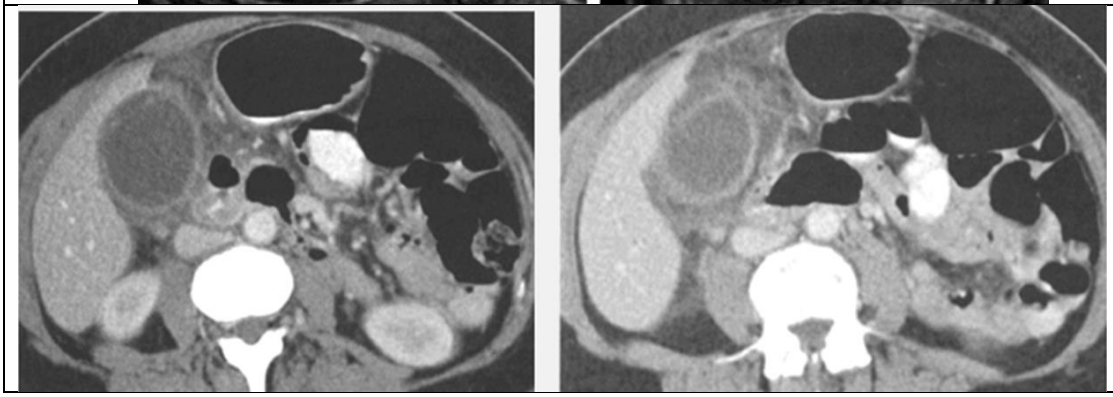


❖Complications of Acute Cholecystitis:

1) Acute Gangrenous Cholecystitis

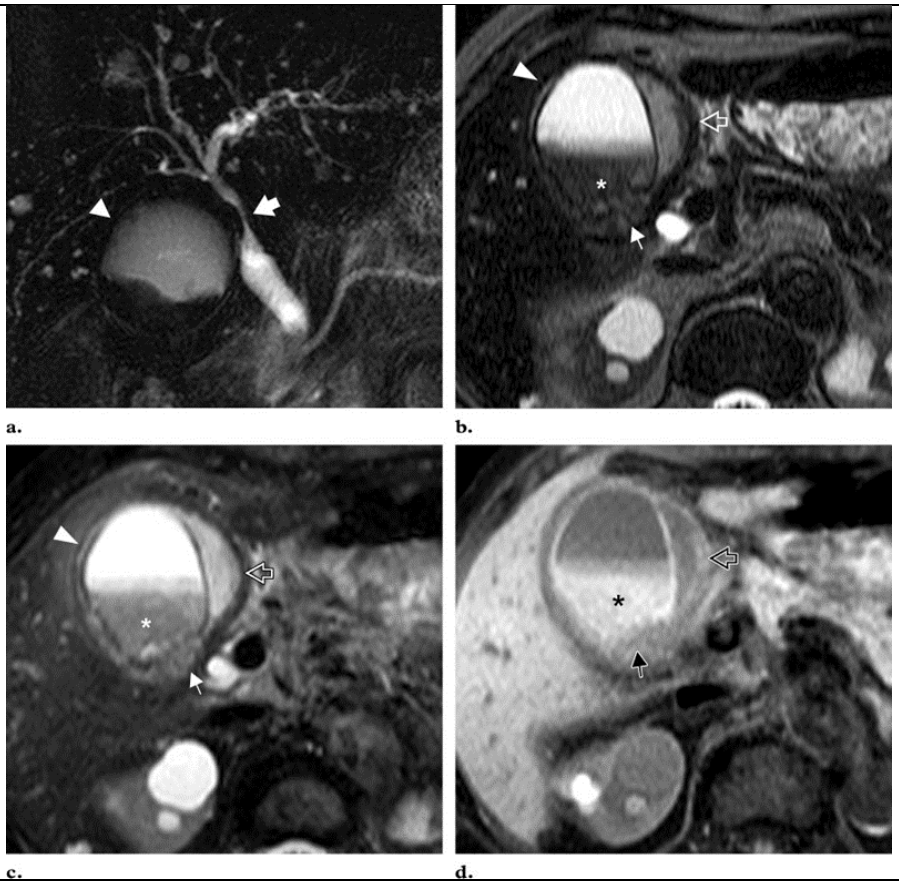
- Asymmetric gallbladder wall thickening due to intramural microabscesses, intramural hemorrhage,
- "Interrupted Rim Sign" = patchy enhancement of the gallbladder mucosa, represents areas of necrosis
- Complex pericholecystic fluid collections containing debris.
- Adherent omentum



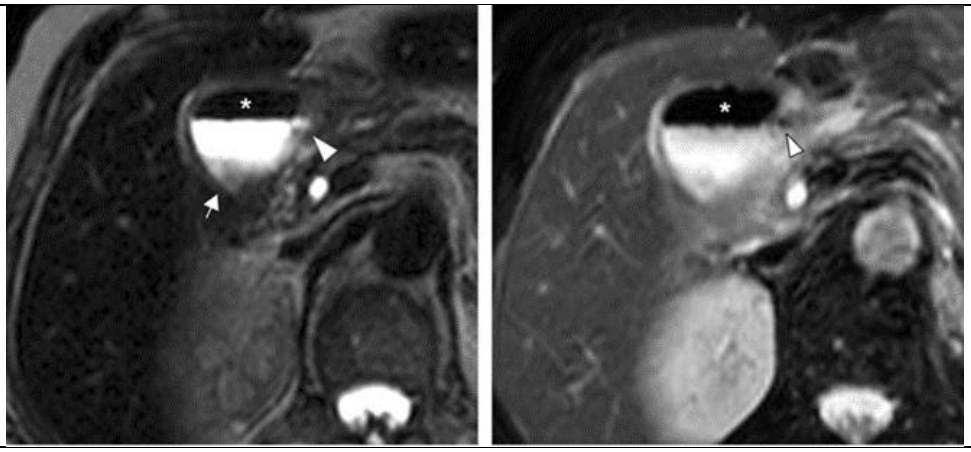


2) Gallbladder Perforation with Pericholecystic Abscesses

- Localized fluid collections with rim enhancement.

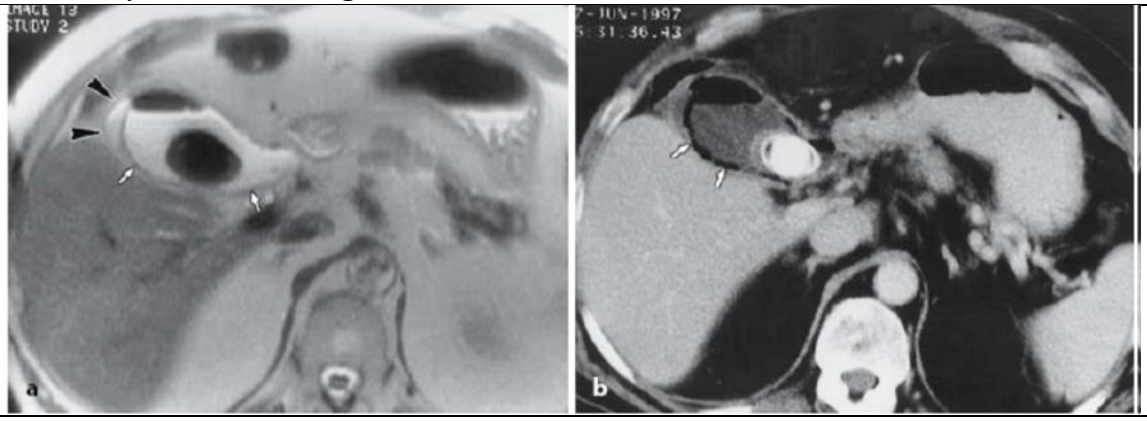


3) Gallbladder Perforation with Enterobiliary Fistula



4) Emphysematous Cholecystitis

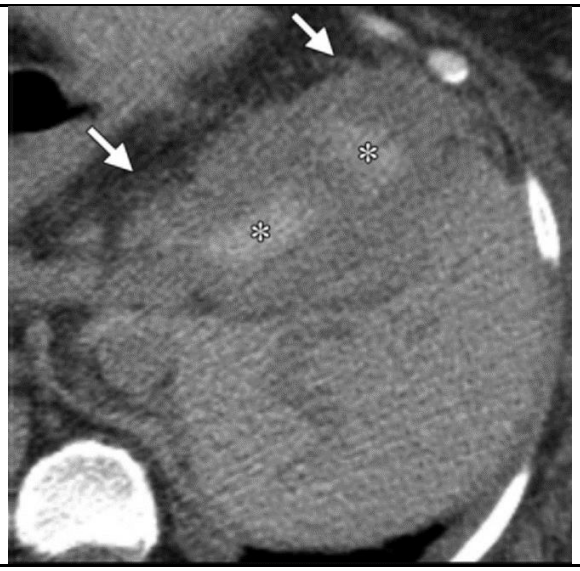
- Emphysematous cholecystitis is caused by gas forming bacteria that infect the gallbladder wall and produce intramural and intraluminal gas.
- Can occur in diabetic patients or in cases of atherosclerosis of the cystic artery with resulting ischemia



5) Hemorrhagic Cholecystitis

- A rare complication of acute calculous or acalculous cholecystitis
- may be caused by trauma, anticoagulation, malignancy, or iatrogenic or other causes

- **Hyperattenuating bile with fluid-fluid level**



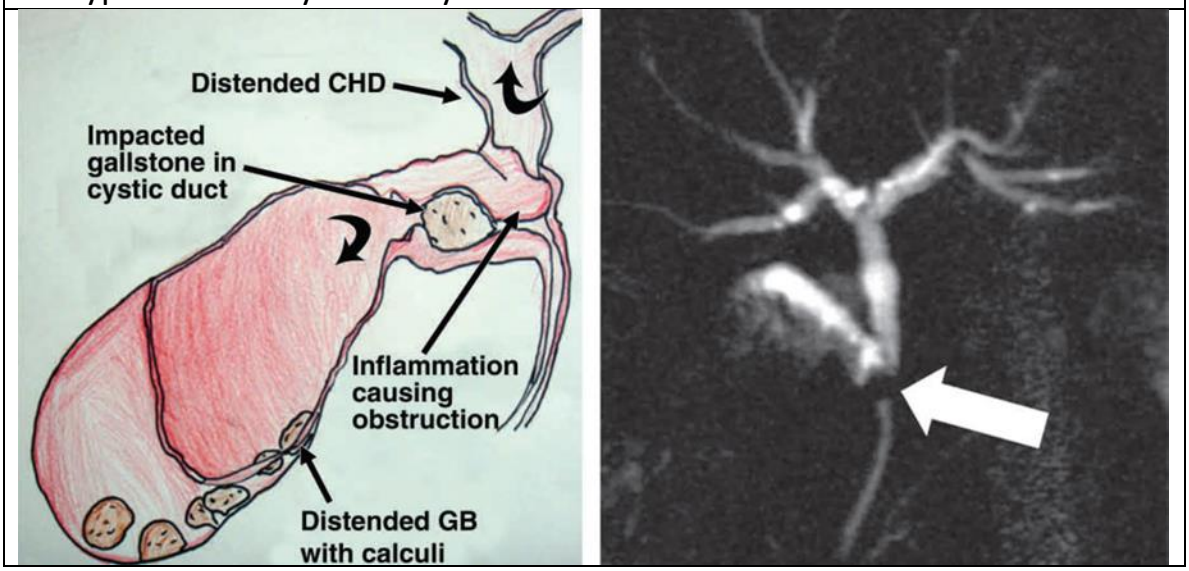
6) MIRIZZI SYNDROME

- A rare complication of gallstone disease that is caused by an impacted stone in the neck of the gallbladder or the cystic duct, leading to extrinsic compression and subsequent obstruction of the CHD

Imaging findings:

- Impacted gallstone in the cystic duct or gallbladder neck
- Dilatation of the intrahepatic bile ducts, with the level of obstruction at the junction of the cystic duct and CHD.
- Normal distal CBD.
- Acute cholecystitis with pericholecystic inflammatory changes

- Type I: Extrinsic compression of CHD due to cystic duct stone
- Type II: Cholecystobiliary fistula with < 1/3 of ductal wall circumference
- Type III: Cholecystobiliary fistula with 2/3 of ductal wall circumference
- Type IV: Cholecystobiliary fistula with entire ductal wall circumference

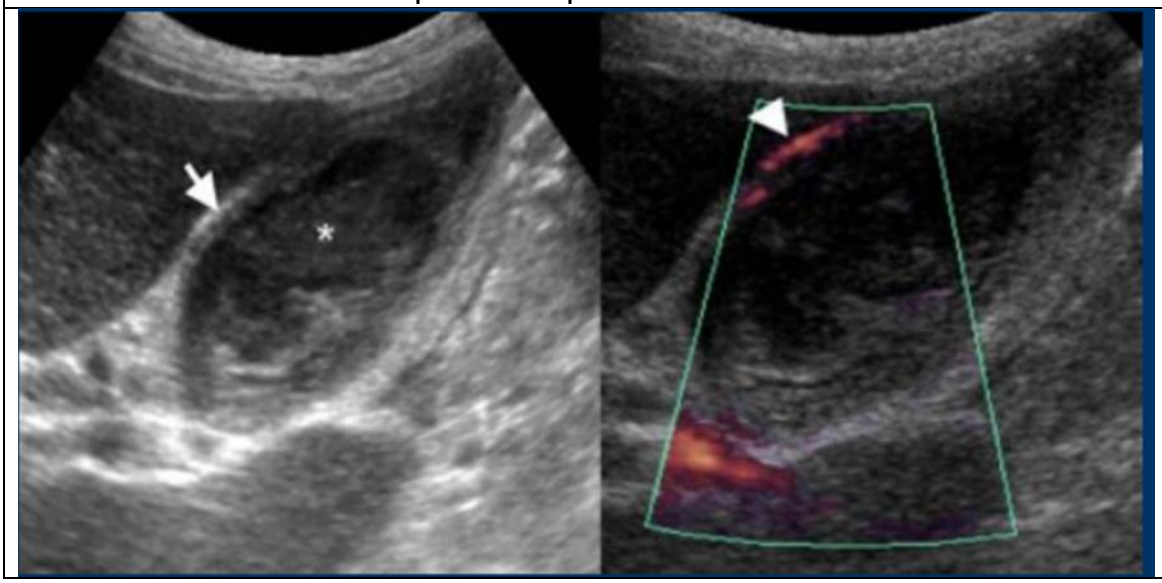


B- Acute Acalculous Cholecystitis

- mainly occurs in critically ill patients, presumably due to increased bile viscosity from fasting and medication that causes cholestasis.
- The imaging features are those of acute cholecystitis, except for the absence of stones whereas gallbladder sludge is usually present

Predisposing Factors:

- Trauma – Burns – Surgery
- Long term Fasting – TPN
- Sepsis
- DM – Renal failure
- Atherosclerotic disease – Systemic vasculitis
- AIDS – Immunocompromised patients

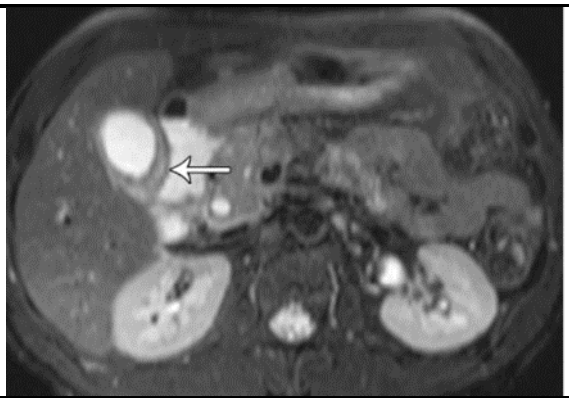


C- Chronic Cholecystitis

Chronic cholecystitis is the most common form of clinically symptomatic gallbladder disease and is almost invariably associated with gallstones.

Imaging findings:

- Gallbladder appears small and contracted
- Non-obstructive stones
- +C: gallbladder wall enhances less intensely than in acute cholecystitis.

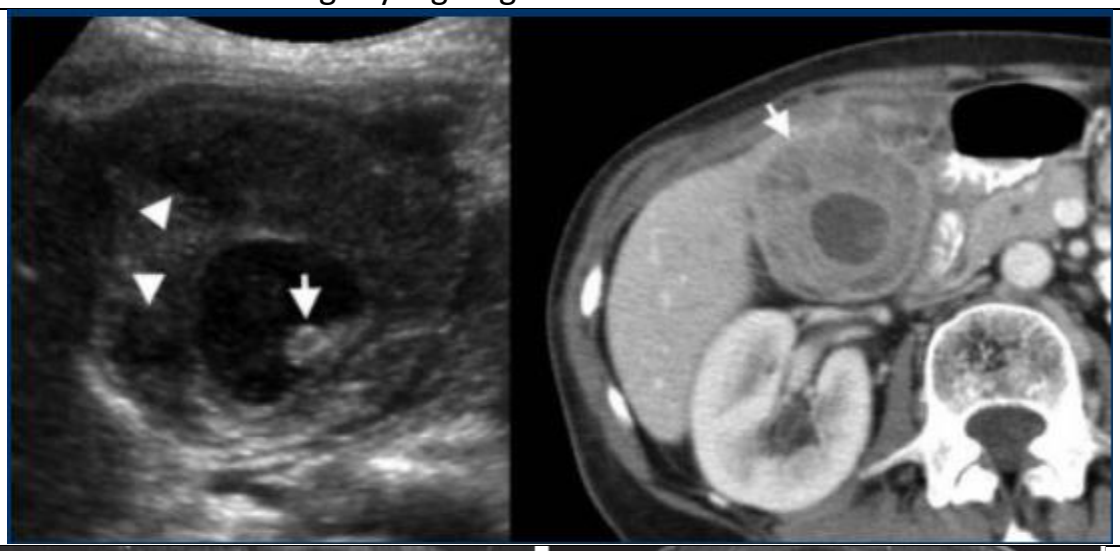


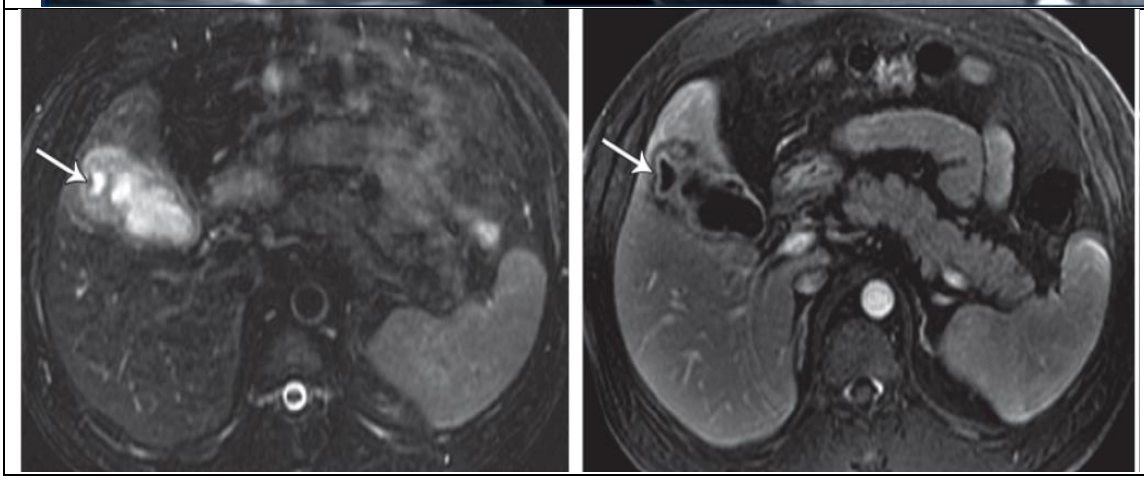
D- Xanthogranulomatous Cholecystitis

- Chronic inflammatory disorder of the gallbladder characterized by a lipid-laden inflammatory process

Imaging findings:

- Focal or diffuse marked wall thickening
- Intramural xanthogranulomatous nodules:
  - US: Hypoechoic
  - CT: hypoattenuating
  - T1: high signal intensity (fat)
  - T2: iso- to slightly high signal





E- Adenomyomatosis

- Adenomyomatosis is characterized by excessive proliferation of surface epithelium with deep and branching invaginations (Rokitansky-Aschoff sinuses) into the thickened tunica muscularis.

3 forms:

- **Localized (or fundal) (Most common)**
  - Localized adenomyomatosis is the most common and manifests as a well-formed mass in the gallbladder fundus.
- **Segmental**
  - Segmental adenomyomatosis is characterizedby focal circumferential wall thickening in the midportion ("waist") of the gallbladder wall, producing an "hourglass" appearance
- **Diffuse**
  - Diffuse adenomyomatosis is characterized by diffuse wall thickening with intramural diverticula that appear as cystic spaces within the wall.

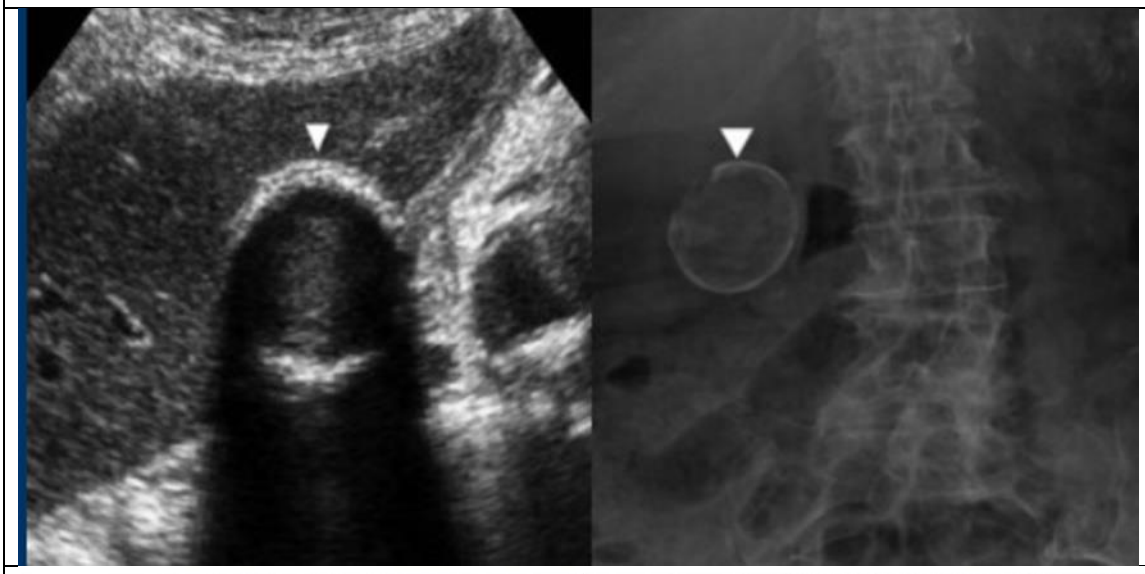
Imaging findings:

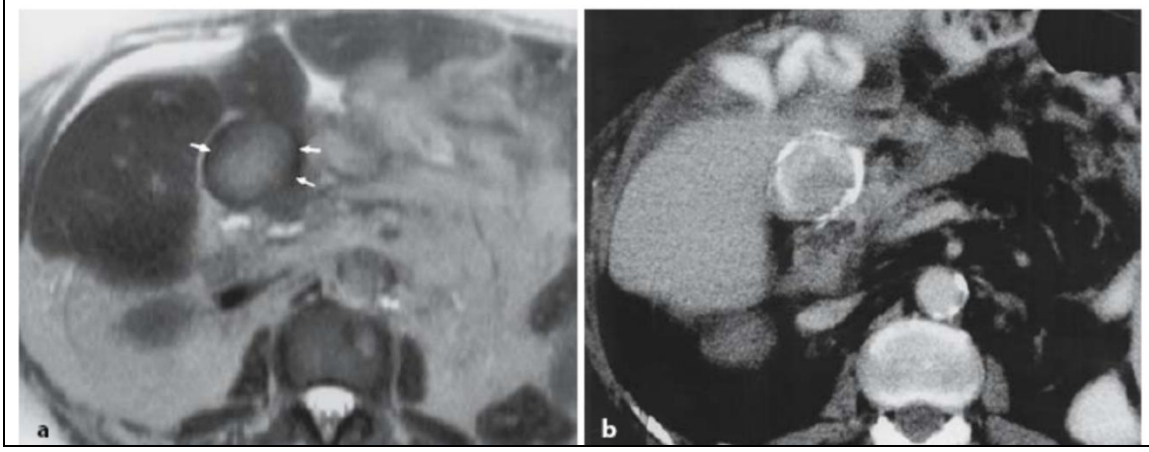
- Focal or diffuse gallbladder wall thickening.
- US hallmark => "comet tail" reverberation artifact that is seen emanating from small echogenic foci in the gallbladder wall.
- MRCP => the Rokitansky- Aschoff sinuses manifest as "pearl necklace gallbladder" a finding that is typical of adenomyomatosis

GB
fundus
Liver
Liver

F- Porcelain Gall Bladder

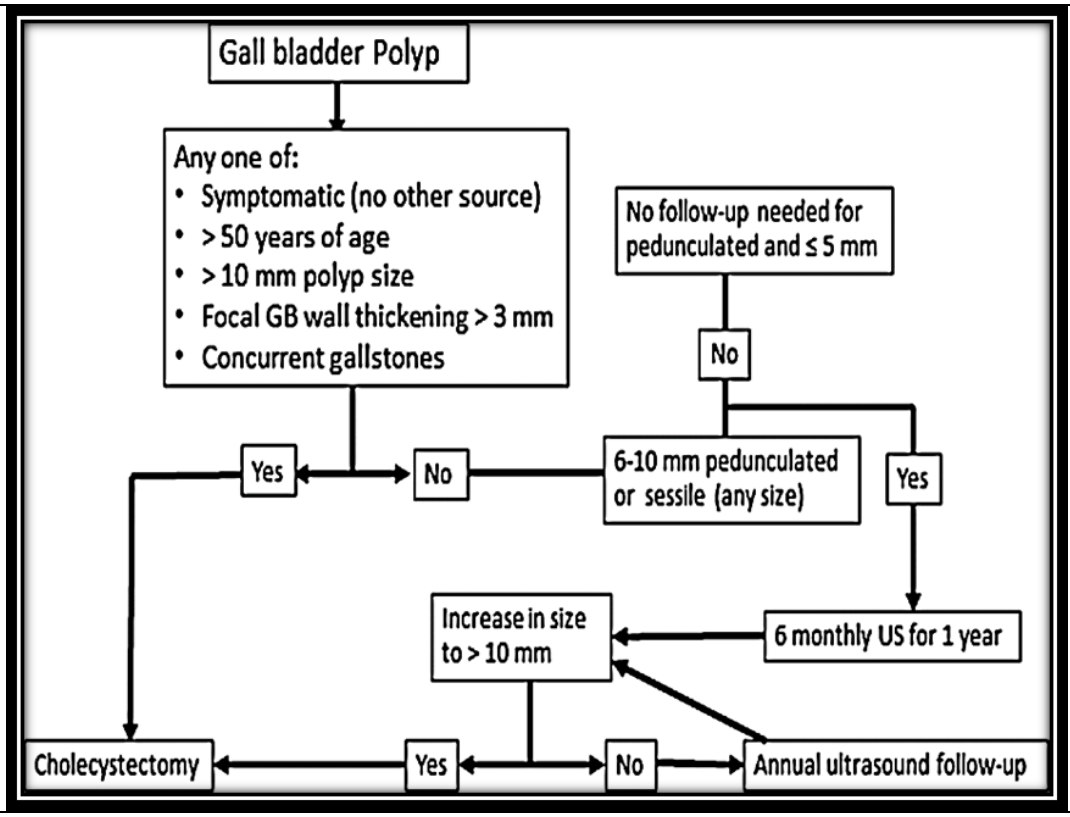
- Chronic cholecystitis characterized by mural calcification
- Calcification in muscularis mucosa
- Risk factor for GB carcinoma (10-30%) (weak association)





Gall Bladder Polyps

Benign Polyps	Malignant Polyps
• 95% of all polyps • Cholesterol polyps : > 50% • Adenoma: ~ 30% • Inflammatory polyps • Adenomyomatosis	• 5% of all polyps • Adenocarcinoma : ~ 90% of malignant polyps • Other rare entities including • Metastases to gallbladder • Squamous cell carcinoma • Angiosarcoma

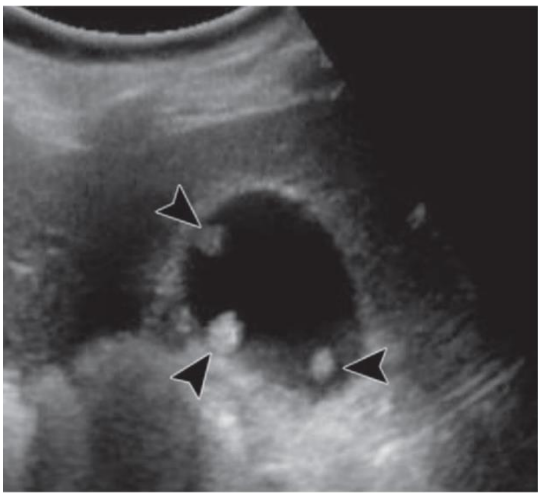


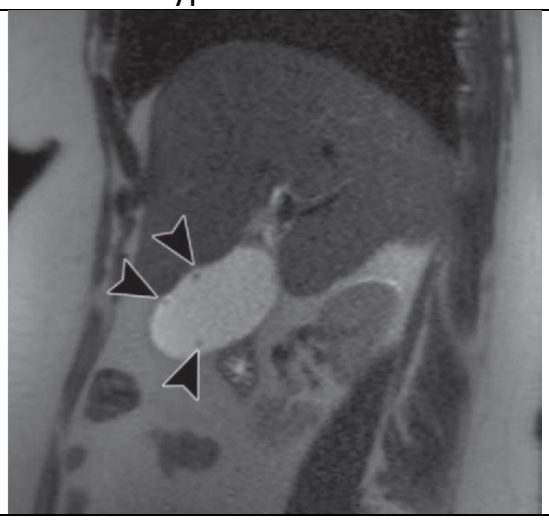
A- Benign GB Polys

1) Cholesterol Polyps

90% of GB polyps

- Small rounded intraluminal polyps
- Attached to gall bladder wall.
- No acoustic shadow
- Small size
  - 90% are less than 10 mm
  - most are less than 5 mm
- Morphology:
  - Small polyps => adherent to the wall and a smooth
  - Larger lesions => pedunculated and granular in outline
- Echogenicity varies with size
  - Small polyps are echogenic but non-shadowing
  - Larger cholesterol polyps tend to be hypoechoic

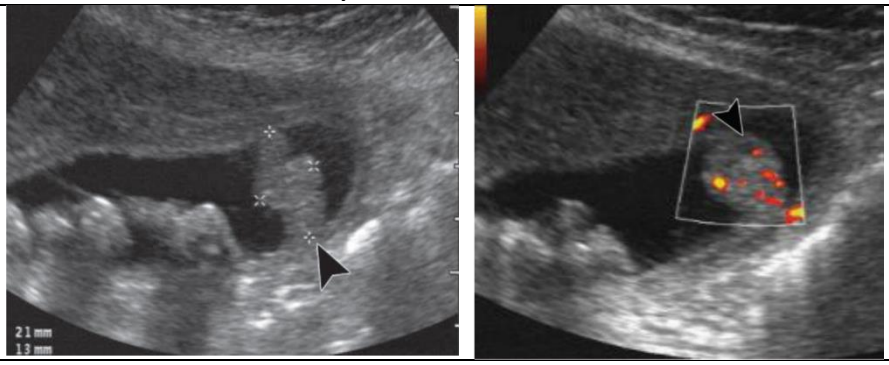




2) Inflammatory Polyps

- 10% of all gallbladder polyps.
- 2ry to gallstones and chronic inflammation.
- US: nonspecific appearance

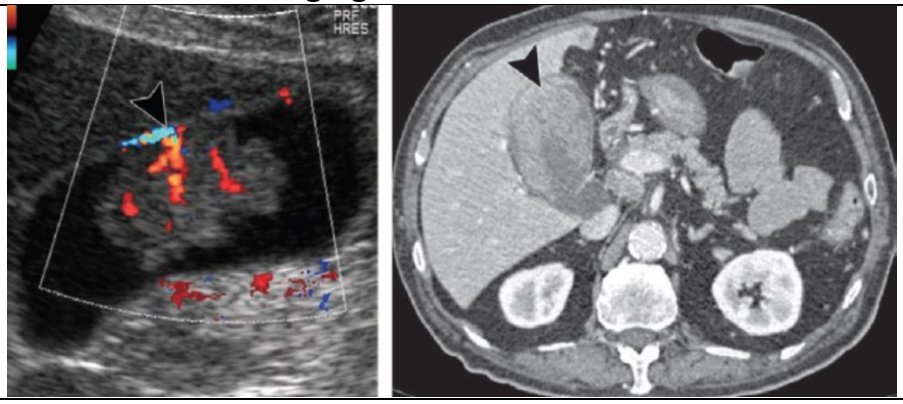
- Polypoid intraluminal gallbladder mass (arrowhead)
- Duplex: internal vascularity



3) Gallbladder Adenoma

- Rare
- 4%–7% of all gallbladder polyps
- Seen in patients with primary sclerosing cholangitis (PSC) and GIT polyposis syndromes, such as Peutz-Jegher and Gardner syndromes.

- Solitary
- vary in size (up to 20 mm)
- Sessile or pedunculated
- Doppler: Internal vascularity
- CT/MRI: Enhancement similar to that of adenocarcinoma
- Adenomas cannot be differentiated from polypoid GB adenocarcinoma at imaging



B- Malignant GB Polyps

1) GB Carcinoma

- 5th most common GIT malignancy worldwide.
- F > M
- It is more commonly seen in the elderly.
- Very poor prognosis because it is often detected at an advanced stage

Associations & Risk Factors:

- Gallstones / Chronic cholecystitis
- GB wall calcification (Porcelain GB)
- Primary sclerosing cholangitis (PSC)
- Anomalous pancreaticobiliary ductal union
- Most types of choledochal cysts
- Ulcerative colitis; rarely Crohn disease
- Familial polyposis coli

Pathological Types

- Adenocarcinoma (most common)
  - Papillary form
  - Signet ring
  - Mucinous
- Adenosquamous carcinoma
- Small cell carcinoma
- Sarcomas

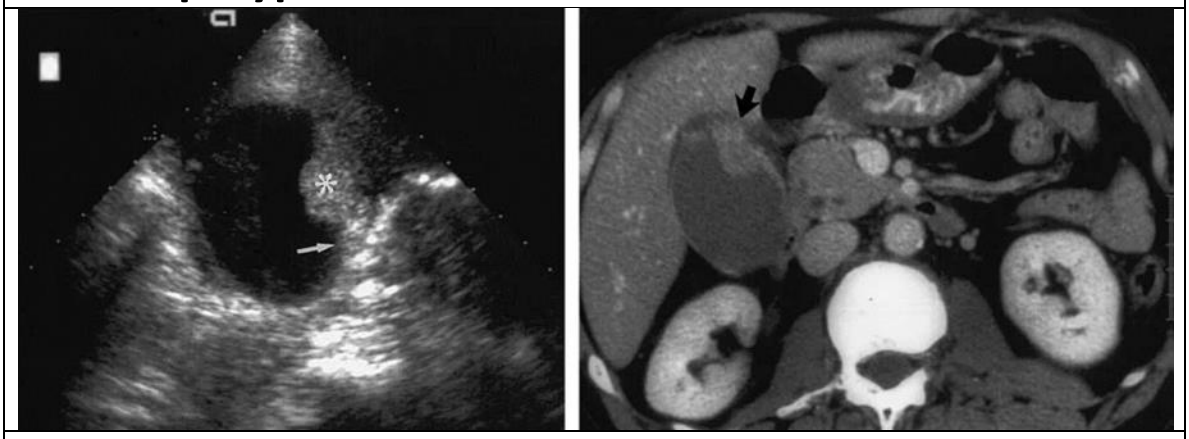
3 Morphological Forms

- **Mass in the GB fossa**
- **Irregular eccentric GB wall thickening >3mm**
- **Intraluminal solitary polyp >1cm**

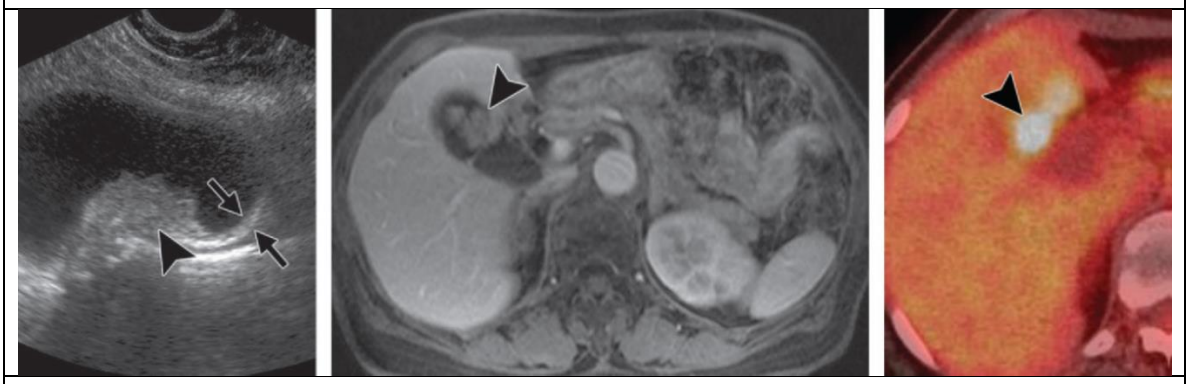
Extensions:

- Hypovascular mass infiltrating GB fossa, invading liver along main lobar fissure and portahepatis.
- Nodal metastasis to porta hepatis and peripancreatic LNs

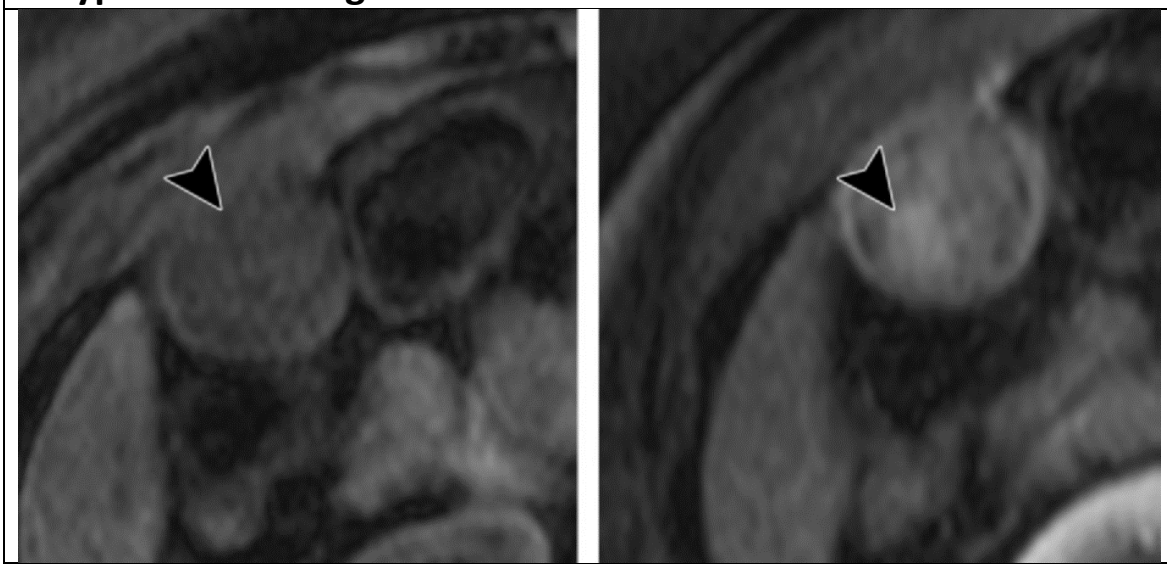
Sessile polyp



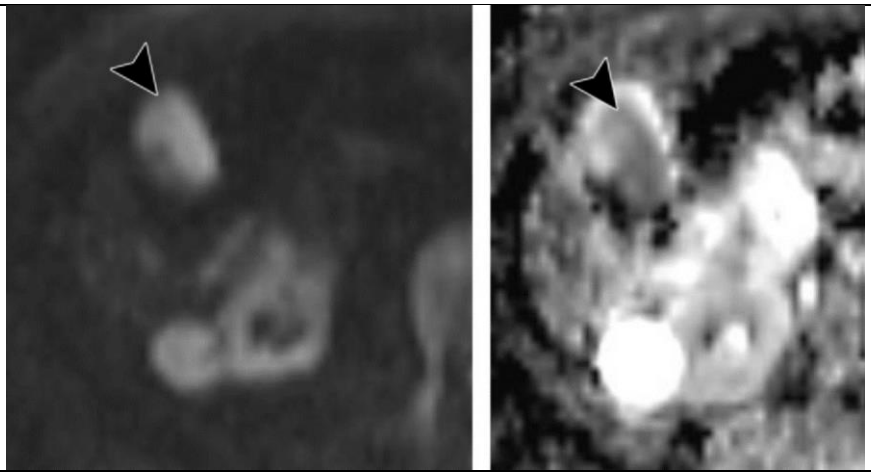
Frondlike Mass



Polypoid enhancing GB mass



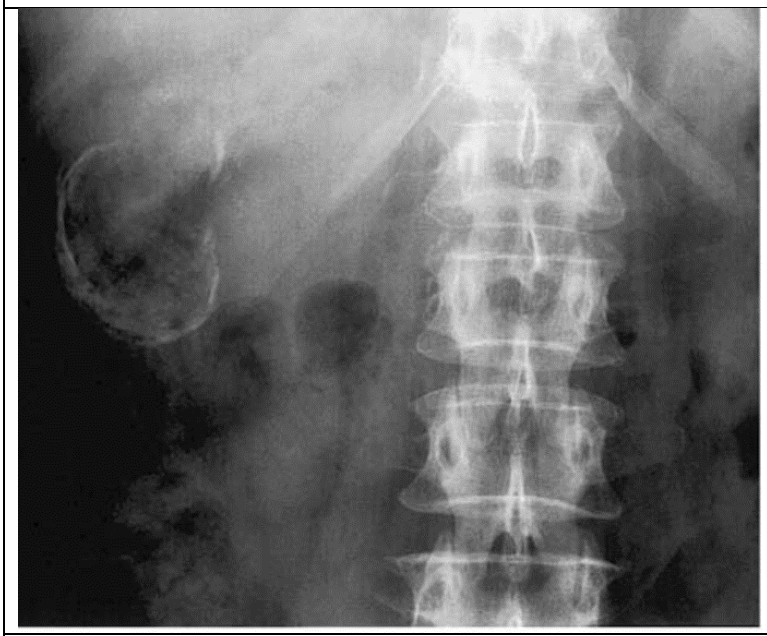
GB Mass => Restricted DWI



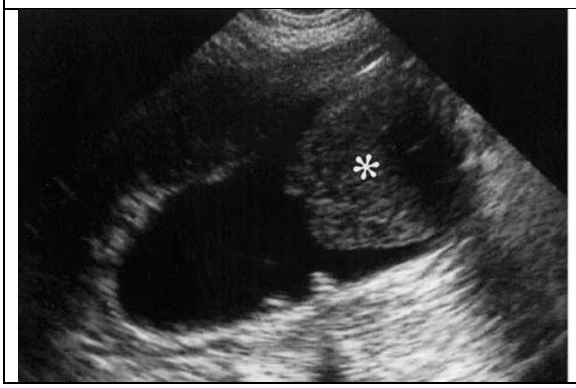
GB mass => Invading liver and portahepatis

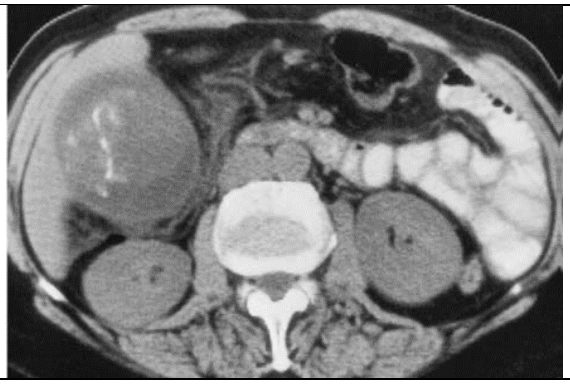
GB mass + Portahepatis LNs

Porcelain GB + GB Mass



GB Stones + GB Mass:

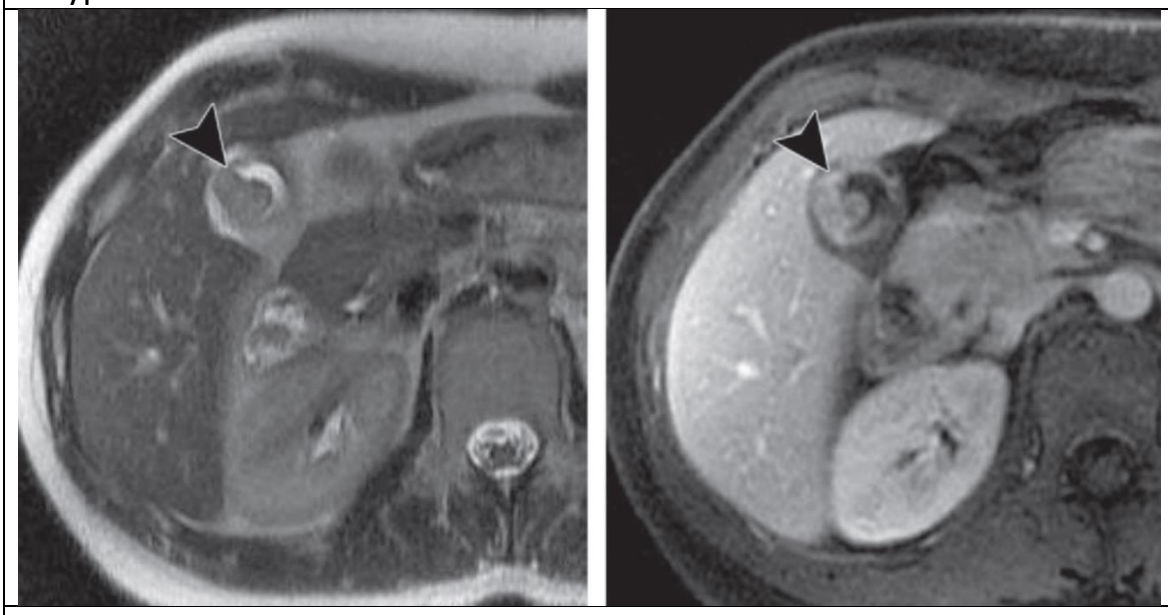




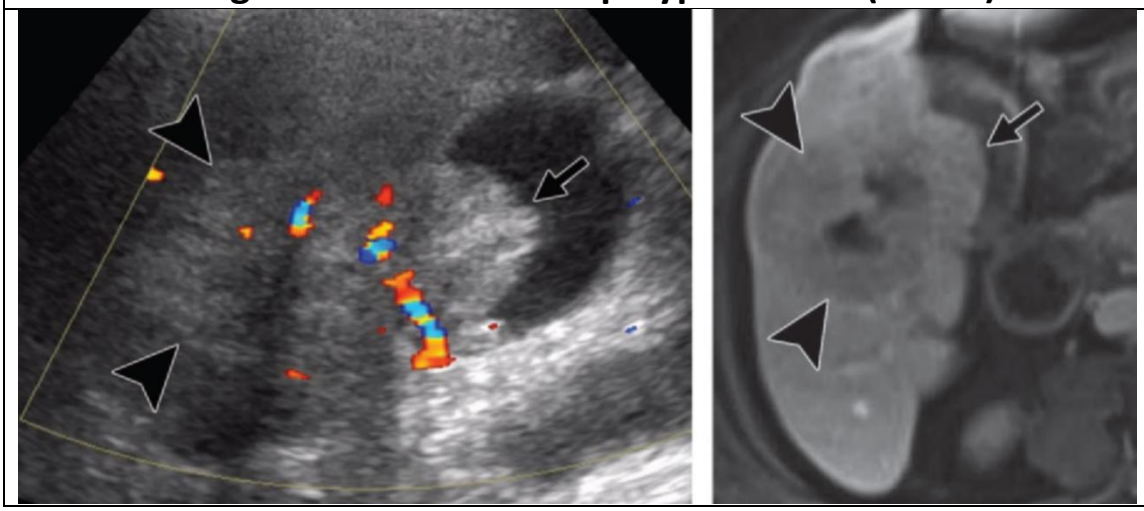
2) GB Metastasis

- The most common primary tumor to metastasize to GB is melanoma (60% of all GB metastases)
- Cancers of the kidney and lung may hematogenously spread to GB.
- Hepatocellular carcinoma and cholangiocarcinomamay directly invade the gallbladder

Polypoid Melanoma Metastasis



HCC invading GB => intraluminal polypoid mass (arrow).



Biliary Dilatation

Obstructive	Non-Obstructive
A - Biliary Stricture	
Benign stricture: **i) Iatrogenic** • Cholecystectomy • Liver transplantation **ii) Post – Inflammatory:** • Post Radiation • Post Chemotherapy • Post Pancreatitis **iii) Infection / Inflammatory** • Acute cholangitis = Ascending cholangitis • Recurrent pyogenic cholangitis = Oriental cholangitis • Primary sclerosing cholangitis • IgG-4 sclerosing cholangitis • Eosinophillic cholangitis • AIDS cholangiopathy • Parasitic Cholangitis	Caroli disease Choledochal cyst
Malignant Stricture: • Cholangiocarcinoma • Pancreatic adenocarcinoma • Ampullary and periampullary carcinoma	
B- Stones = Choledocholithiasis	
C- Compression: • Mirrizi syndrome • Porta-hepatis LNs • Malignant hepatic hilar masses	

A- Benign Biliary Stricture

i- Iatrogenic

Most common cause of a benign biliary stricture is hepatobiliary surgery (80%–90% of cases)

1) Cholecystectomy

- Most commonly => extrahepatic bile duct stricture

MRCP:

- Short-segment smooth extrahepatic bile duct stricture at
- Junction of cystic duct with CHD
- Confluence of left and right hepatic ducts

MRI

- Narrowed segment commonly demonstrates a thin, nonenhancing wall with smooth margins

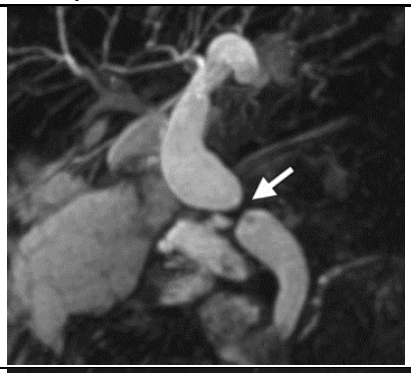
2) Liver Transplantation:

Biliary strictures may develop in

- 5%–15% of cadaveric liver transplants
- 28%–32% of living donor liver transplants

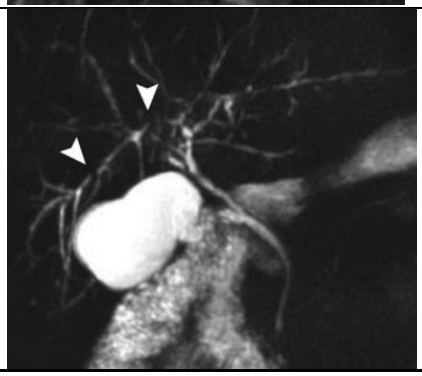
Anastomotic Stricture:

- due to technical factors and local ischemia
- Single, short stricture segment
- Localized at biliary duct–duct anastomosis (arrow)



Nonanastomotic Strictures

- Secondary to ischemic (hepatic artery thrombus or stenosis
- Multiple strictures involve long segments, of intrahepatic bile ducts proximal to the anastomotic site.



ii- Post – Inflammatory:

❖Post – Pancreatitis

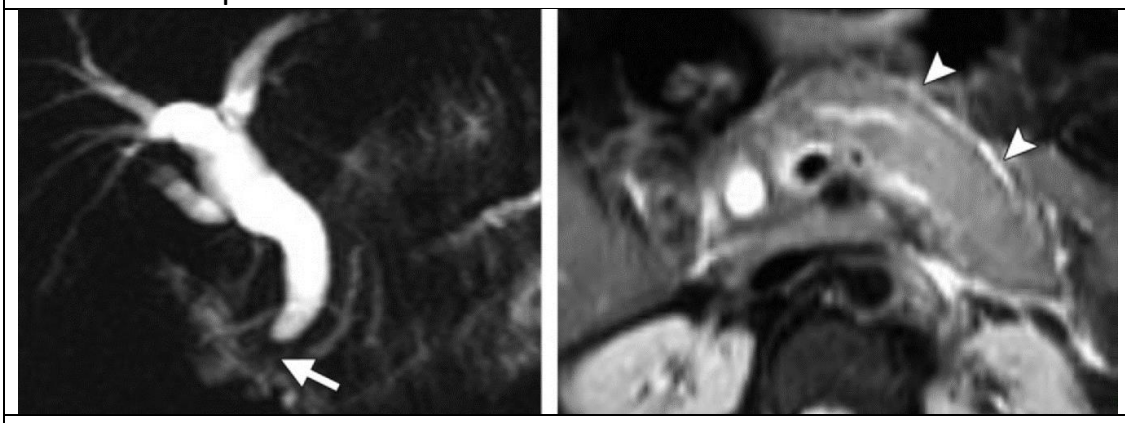
- Chronic pancreatitis accounts for about 10% of all benign biliary strictures.
- The intrapancreatic portion of the CBD is most commonly involved due to fibrosis of the periductal pancreatic parenchyma

MRCP:

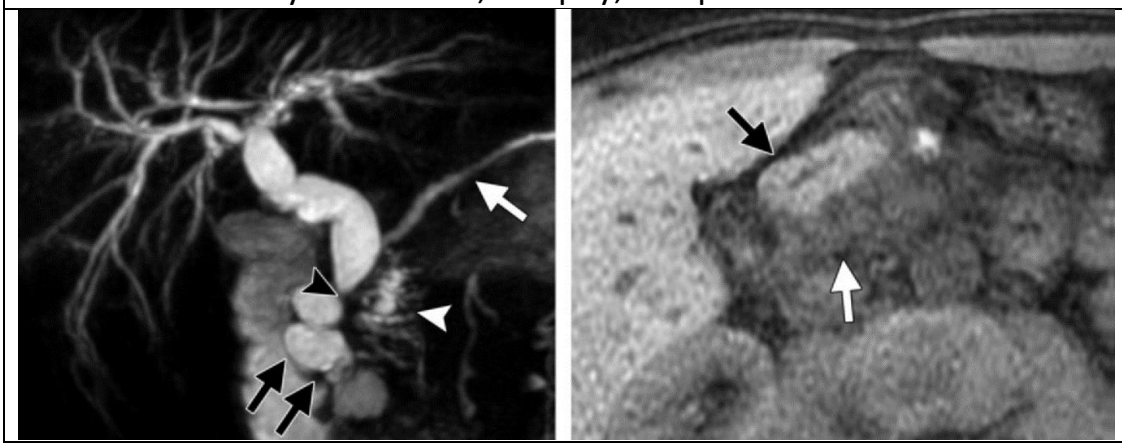
- Smooth stricture in **distal CBD** with gradual tapering.

MRI:

- **Acute pancreatitis**
  - Enlarged pancreas, peripancreatic fat stranding
  - Peripancreatic fluid collections



- **Chronic pancreatitis**
  - Parenchymal fibrosis, atrophy, and pancreatic duct dilatation



iii- Infectious / Inflammatory = Cholangitis

1) Bacterial Acute Cholangitis = Ascending Cholangitis

- Inflammation of intra-/extrahepatic bile duct walls
- Usually due to ductal obstruction and infection.
- Charcot triad = pain + Fever + Jaundice

Obstruction of CBD:

- Stones is the most common cause.
- Instrumentation of the biliary tree
- Malignant disease
- Sclerosing cholangitis

Complications:

Acute complications of BAC:

- Sepsis.
- hepatic abscesses
- Portal vein thrombosis
- Bile peritonitis.

Chronic complications of BAC

- Portal vein thrombosis
- Biliary stricture
- Sclerosing cholangitis

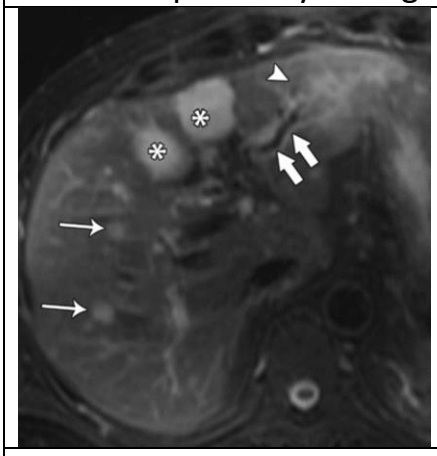
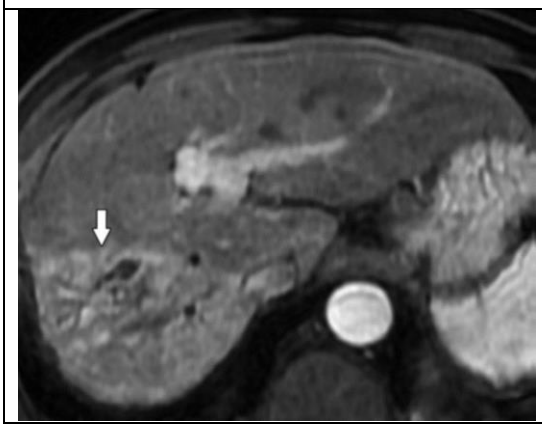
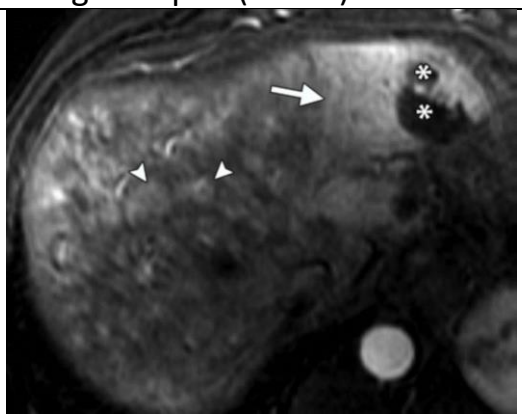
Imaging Findings:

Biliary changes:

- Dilatation of intrahepatic biliary ducts.
- Wall thickening and enhancement of intrahepatic biliary ducts.
- Pneumobilia

Parenchymal changes:

- Increased T2 signal + contrast enhancment => wedge-shaped or peribiliary distribution.
- Liver abscesses
- PV thrombosis

MRCP: Dilated IHBRS	Enhancement of the walls of IHBRS
T2: Patchy (thin arrows) and wedge shaped (arrowhead) areas of increased parenchymal signal	
	
T1+C FS: segmental parenchymal enhancement (arrow).	T1+C FS: parenchymal enhancement in a peribiliary (arrowheads) and wedge-shaped (arrow) distribution.
	

2) Recurrent Pyogenic Cholangitis (RPC) = Oriental Cholangitis

- **Asian patients:**
  - Hepatobiliary infestation with Chlonorchis sinensis (liver fluke)
- **Location:**
  - Lateral segment of the left lobe
  - Posterior segment of the right lobe
- **Cholangiocarcinoma:**
  - tend to occur in atrophied or heavily stone-burdened segments

Imaging Findings:

Biliary changes:

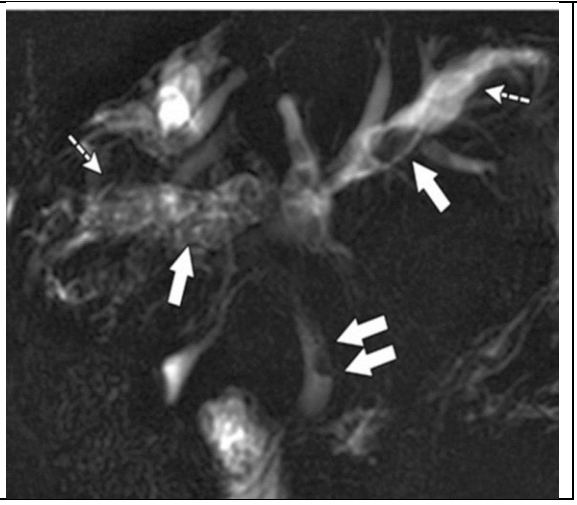
- Strictures of peripheral intrahepatic ducts
- Dilatation of central and extrahepatic bile ducts
- Bile duct wall thickening and enhancement
- Intrahepatic pigment stones without GB stones

Parenchymal Atrophy:

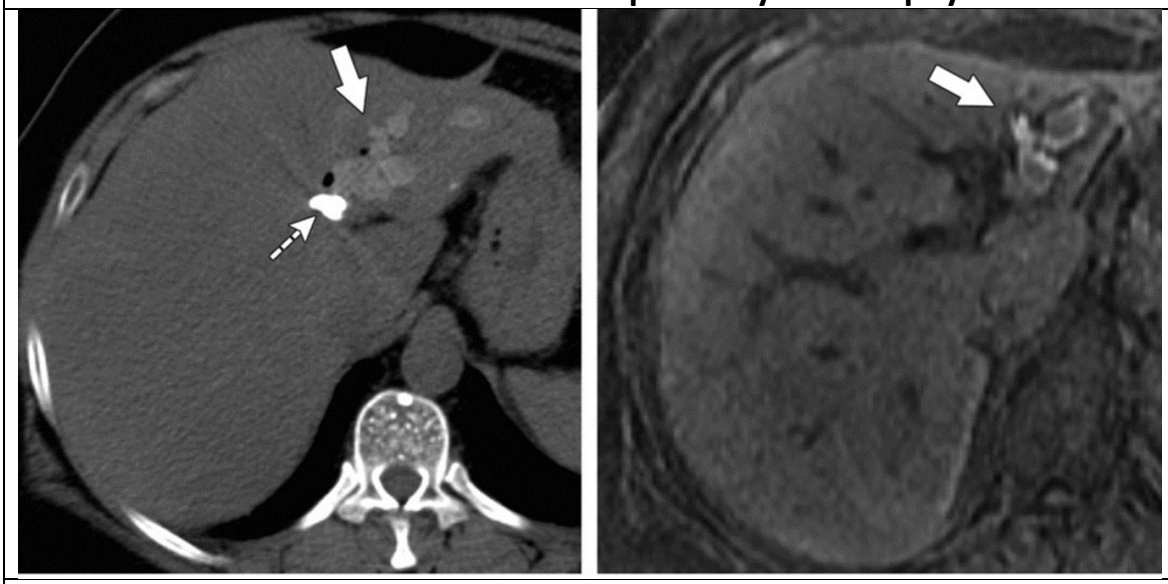
- is common manifestation of the chronic nature of the disease.
- typically affects the left lobe or right posterior segments.

MRCP:

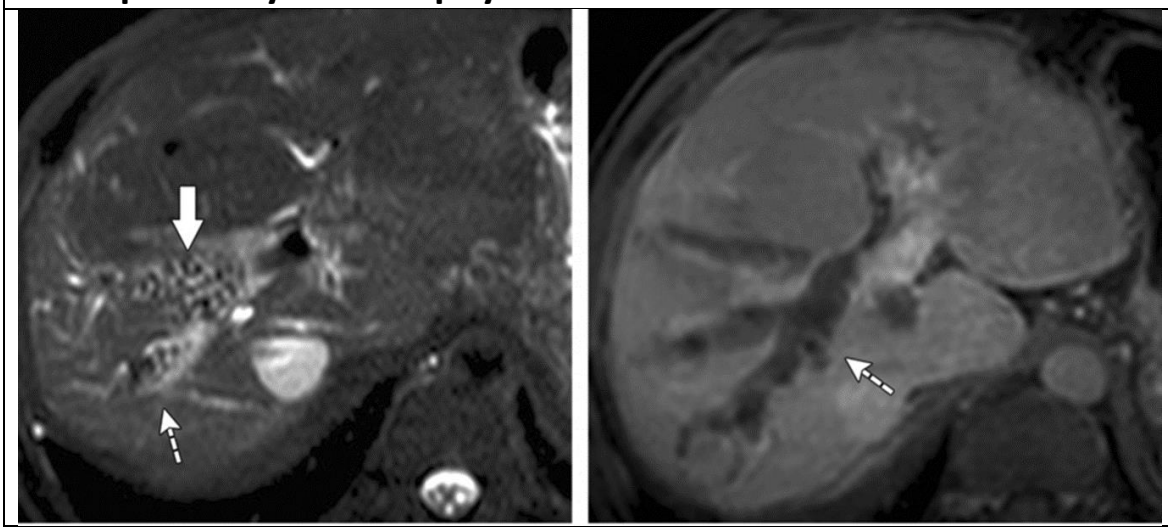
Dilatation of intrahepatic biliary tree (dashed arrows)
Multiple intrahepatic duct stones of different sizes (top solid arrows)
CBD stones (bottom solid arrows).



Left lobe lateral segment:
Dilated IHBRs + Bile duct stones + focal parenchymal atrophy



Right lobe posterior segment
Dilated IHBRs + Bile duct stones
Thickened enhanced bile duct walls
Focal parenchymal atrophy



3) Primary Sclerosing Cholangitis (PSC)

- Primary sclerosis cholangitis (PSC) is an uncommon idiopathic inflammatory condition which affects the biliary tree
- Middle aged males

Associations:

- Inflammatory bowel disease (IBD) (70%):
    - Ulcerative colitis.
- Significantly increased risk of malignancy:
    - Cholangiocarcinoma
    - Colorectal cancer
    - Pancreatic cancer
- Sjogren Syndrome
- Retroperitoneal Fibrosis
- Mediastinal Fibrosis
- Riedel Thyroiditis
- Orbital Pseudotumour

MRCP

- Multifocal short-segment strictures of the intra- and extra-hepatic ducts
- ***Beading***: alternating pattern of strictures and normal or slightly dilated ducts
- ***Pruning:*** distal bile ducts are narrowed
- ***Mural irregularity:*** irregular luminal margin
- ***Diverticula:***
- ***Distortion of the biliary tree*** due to associated cirrhosis

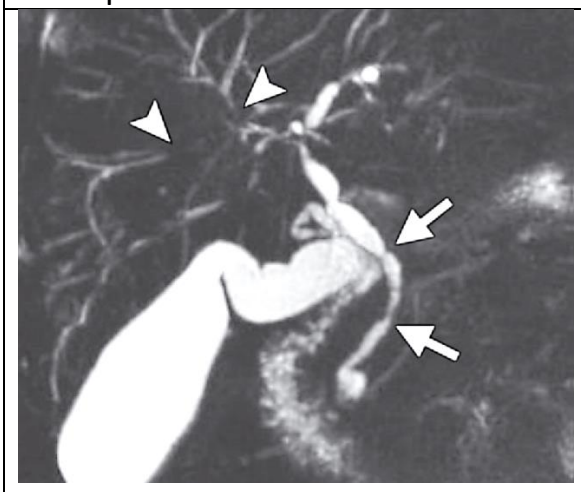
MRI:

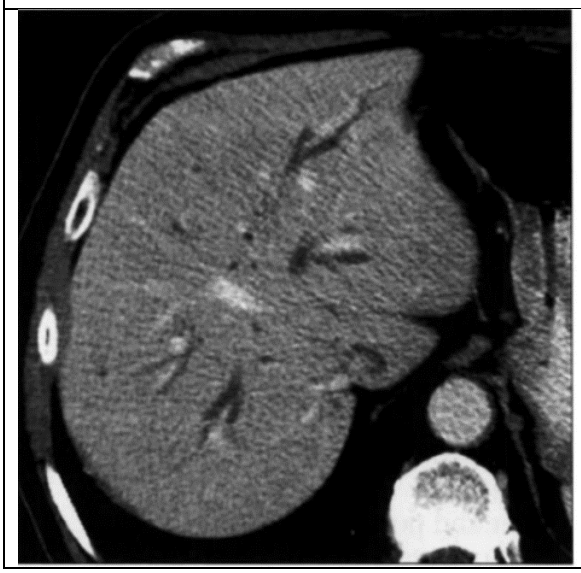
- Multifocal wall thickening and enhancement of the bile ducts
- Multiple enhancing areas of fibrosis in the liver periphery.
- Periportal lymph nodes
- Intrahepatic duct stones may also be seen

Complications:

- Biliary cirrhosis
- Cholangiocarcinoma (6-15%)

MRCP:
Beaded = Multiple strictures + interspersed with focal dilatations



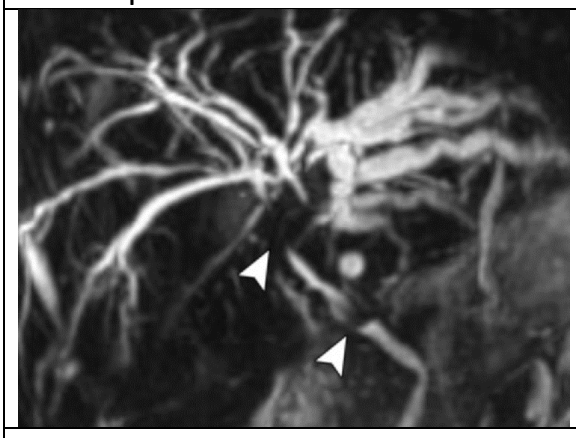
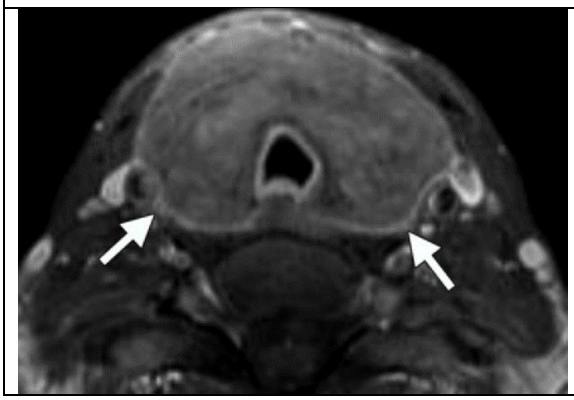
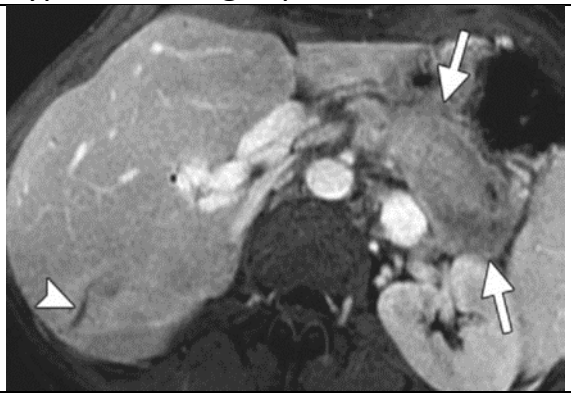


4) IgG4-related Sclerosing Cholangitis

- is the biliary manifestation of IgG4 sclerosing disease

4 patterns of biliary strictures:

(a) stricture of the distal CBD
(b) diffuse strictures of the intra- and extrahepatic bile ducts
(c) hilar stricture and distal CBD stricture
(d) isolated hilar stricture

IgG4-associated sclerosing cholangitis = interval multiple strictures involving the extra- and intrahepatic bile ducts	Retroperitoneal Fibrosis
	
Reidel thyroiditis = Diffusely enlarged thyroid	Autoimmune Pancreatitis = sausage-shaped pancreas with a thick hypoenhancing capsule
	

5) AIDS Cholangiopathy

- AIDS cholangiopathy is a form of secondary sclerosing cholangitis that occurs in AIDS patients with a CD4 count < 100/mm3.

Chronic inflammation of the biliary tract by opportunistic pathogens such as:

- **Cryptosporidium parvum**
- **Cytomegalovirus (CMV)**
- **Mycobacterium avium complex (MAC)**
- **Microsporidia species**
- **Herpes simplex virus (HSV)**

MRCP:

- Multiple intra- and extrahepatic biliary strictures + associated dilatation simulating PSC.
- Papillary stenosis with dilated CBD
- Long-segment (1–2-cm) extrahepatic bile duct stricture

MRI:

- Acalculous cholecystitis
- Wall thickening and enhancement of the bile ducts

6) CHEMOTHERAPY CHOLANGITIS

- Iatrogenic cholangitis following intra-arterial chemotherapy for treatment of primary hepatic tumors and metastases

- Strictures of common hepatic duct are characteristic
- Strictures frequently involve biliary bifurcation.
- Intrahepatic duct strictures are less common.
- Sparing of distal common bile duct; hallmark.
- Gallbladder & cystic duct may be involved.
- Distribution of strictures in biliary tree reflects hepatic arterial supply to bile ducts.

B- Malignant Biliary Stricture

Cholangiocarcinoma

- Cholangiocarcinoma is a primary tumor arising from the bile duct epithelium
- 2nd most common primary malignancy of the liver after HCC.
- Intrahepatic cholangiocarcinoma account for 10%–20% of all primary hepatic tumors.

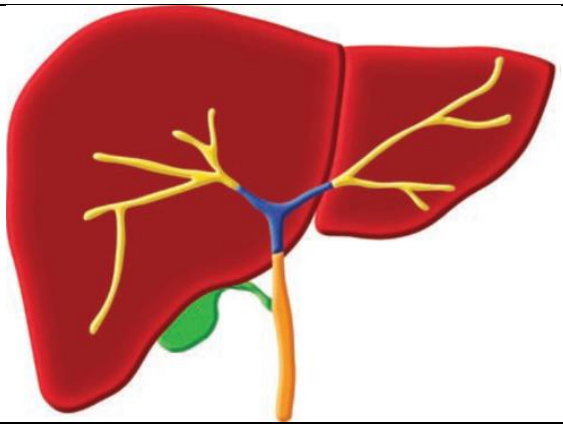
- Age > 65y
- M > F

Risk Factors for Cholangiocarcinoma

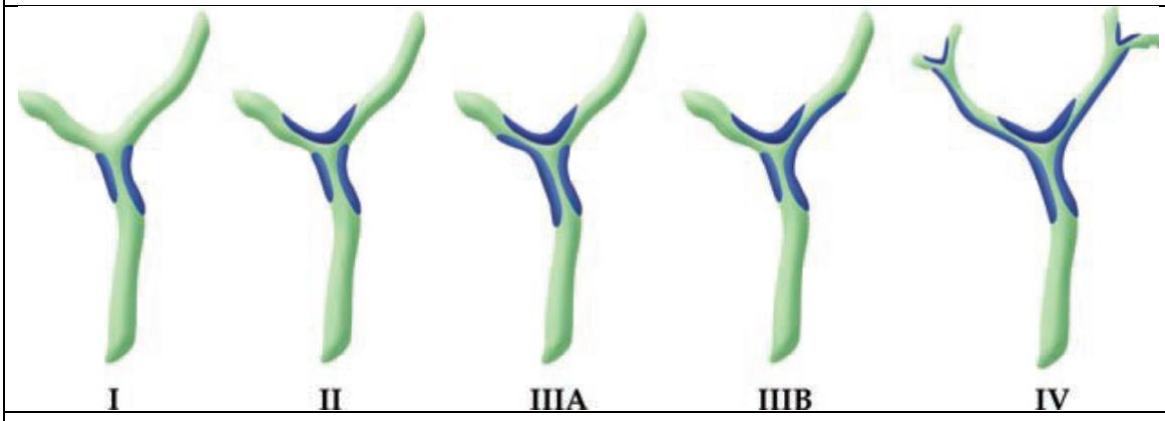
Liver flukes (*Opisthorchis viverini, Clonorchis sinensis*)*
Hepatolithiasis (recurrent pyogenic cholangitis)*
Primary sclerosing cholangitis (PSC)†
Viral infection (human immunodeficiency virus, hepatitis B virus, hepatitis C virus, Epstein-Barr virus)
Anomaly and malformation (anomalous pancreaticobiliary junction and choledochal cyst, fibrocystic liver diseases [eg, Caroli disease])
Environmental or occupational toxin (thorotrast, dioxin, polyvinyl chloride)
Biliary tract–enteric drainage procedures
Heavy alcohol consumption

❖Anatomical Calcification

- **Intrahepatic (peripheral) (yellow)**
- **Perihilar (Klatskin) (blue)**
- **Extrahepatic (orange)**



❖Bismuth and Corlette Classification of Klatskin Tumors



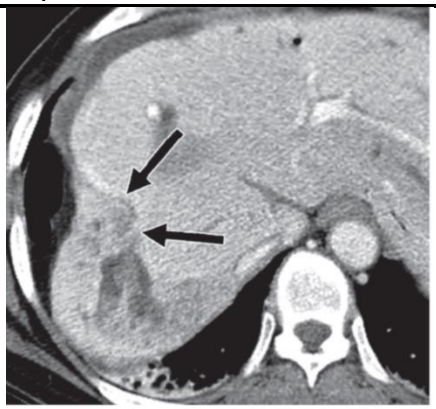
Type I = CHD
Type II = CHD + junction of RHD and LHD
Type IIIA = CHD + biliary junction + RHD
Type IIIB = CHD + biliary junction + LHD
Type IV = CHD + biliary junction + extension to both the RHD and LHD or a multifocal bile duct tumor.

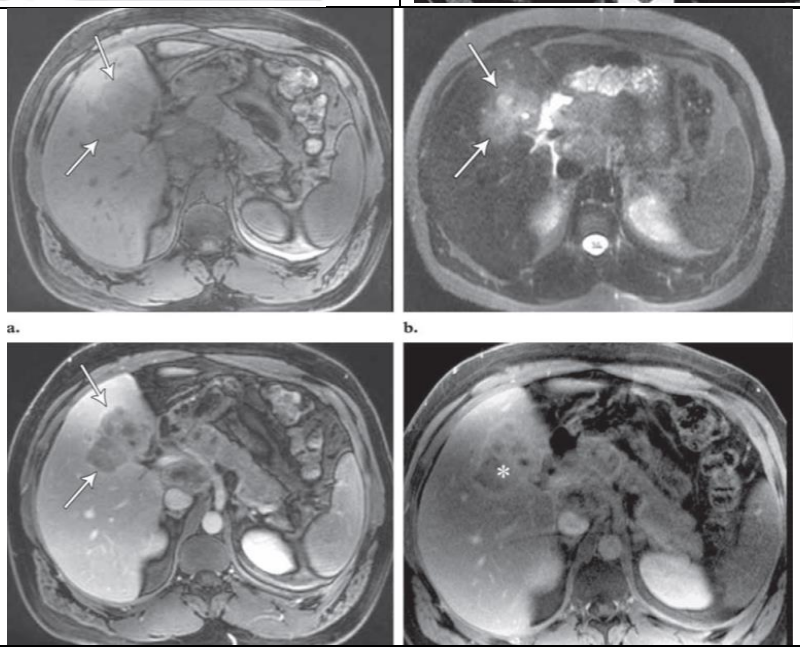
❖Morphologic Classification

- **Mass-forming**
- **Periductal infiltrating**
- **Intraductal growth types**

1) Mass-forming cholangiocarcinoma

- Well defined irregular shaped lesion
- Homogeneous attenuation/signal
- Early irregular peripheral enhancement with gradual centripetal enhancement
- Delayed central enhancement with peripheral washout.
- Capsular retraction
- Parenchymal atrophy of the involved segment
- Presence of satellite nodules
- vascular encasement without the formation of a grossly visible tumor thrombus
- presence of hepatolithiasis
- Peripheral bile ductal dilatation





2) Periductal Infiltrating Type

- is characterized by growth along a dilated or narrowed bile duct without mass formation
- Manifests as an elongated, spiculated, or branchlike abnormality.
- This type of tumor is rare in intrahepatic cholangiocarcinoma, but most hilar cholangiocarcinomas are of this type.
- In the periphery of the liver, a combination of the periductal and mass-forming types is more common than a purely periductal infiltrating lesion.

Imaging findings:

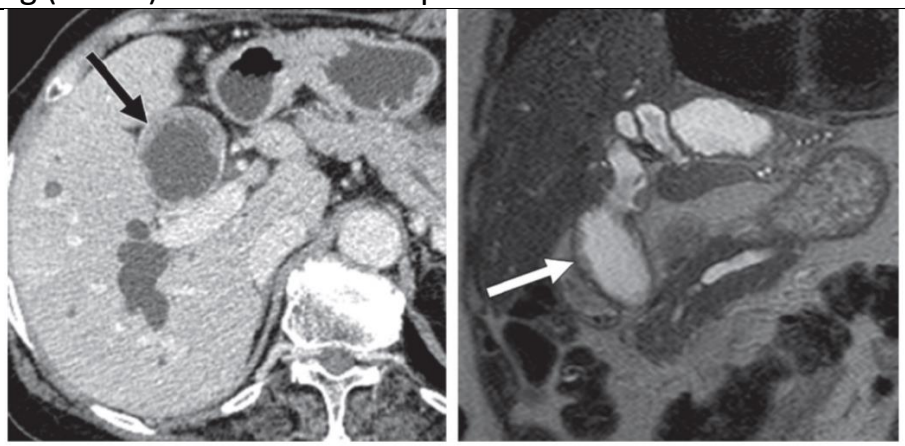
- Diffuse periductal thickening and increased enhancement due to tumor infiltration
- Abnormally dilated or irregularly narrowed duct
- Peripheral ductal dilatation

3) Intraductal Type

Imaging patterns include
(a) diffuse and marked ductectasia + grossly visible papillary mass
(b) diffuse and marked ductectasia + without a visible mass
(c) localized ductal dilatation +intraductal polypoid mass within
(d) intraductal cast like lesions within a mildly dilated duct
(e) a focal stricture-like lesion with mild proximal ductal dilatation

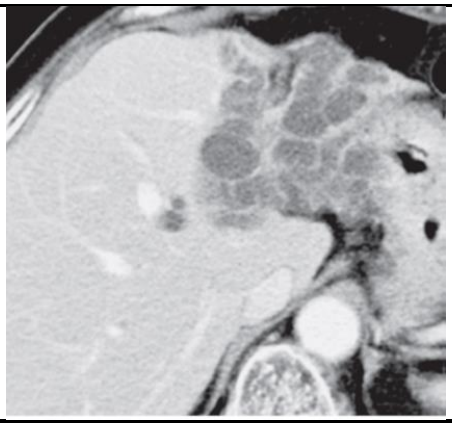
(a) Diffuse and marked ductectasia + grossly visible papillary mass

Markedly dilated intrahepatic duct + mural nodules or irregular wall thickening (arrow) + marked mucin production.



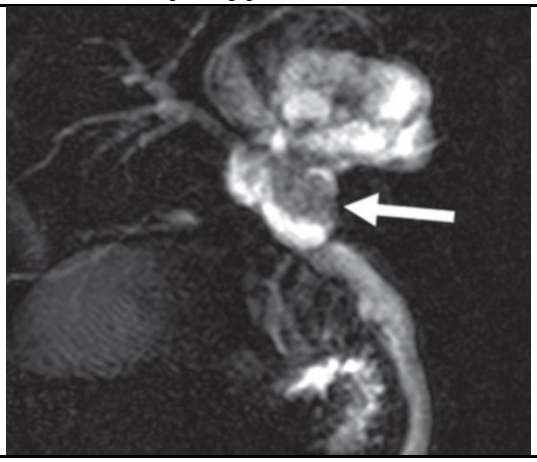
(b) Diffuse and marked ductectasia + without a visible mass

- Diffuse ductal dilatation in the left hepatic lobe through the common bile duct
- No visible intraductal mass



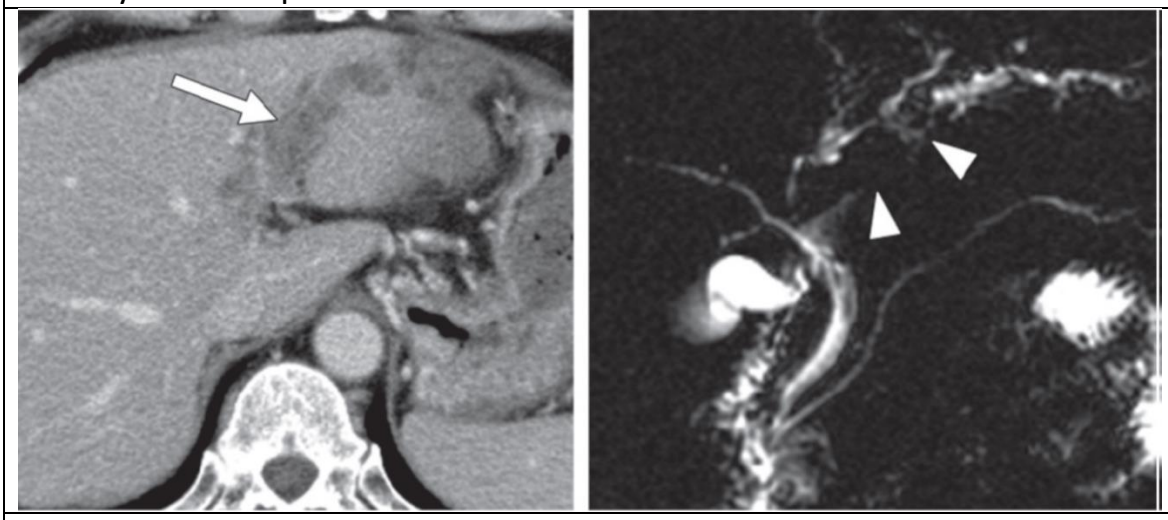
(c) Localized ductal dilatation +intraductal polypoid mass within

- Intraductal polypoid mass
- Localized ductal dilatation (arrow).



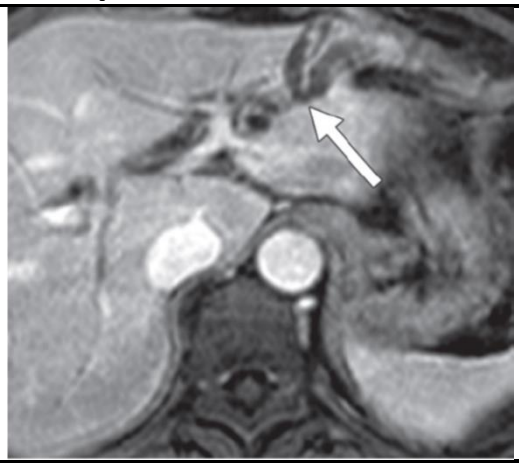
(d) Intraductal cast like lesions within a mildly dilated duct

- Mildly dilated duct with irregularities
- Filled with intraductal soft-tissue material, which may show mild enhancement at CT or MRI
- May mimic impacted stones



(e) Focal stricture-like lesion with mild proximal ductal dilatation

Focal stricture (arrow) with mild ductal dilatation



❖ BilIN

- Microscopic lesions are tumors with flat or micropapillary growth associated with BilIN
- BillN is thought to be a precursor of cholangiocarcinoma in a multistep cholangiocarcinogenesis involving low-grade (BilIN-1) and high-grade (BilIN-2) intraepithelial dysplasia and carcinoma in situ (BilIN-3) .
- BilIN is regarded as a counterpart of pancreatic intraepithelial neoplasia.

Classification of BilIN

Classification	Description
BilIN-1	Low-grade dysplasia of biliary epithelium, mild cellular-nuclear atypia suggestive of neoplasia
BilIN-2	High-grade dysplasia of biliary epithelium, cellular-nuclear atypia evident but insufficient to represent overt malignancy
BilIN-3	Carcinoma in situ, cellular-nuclear atypia representing overt malignancy

C- Choledocholithiasis

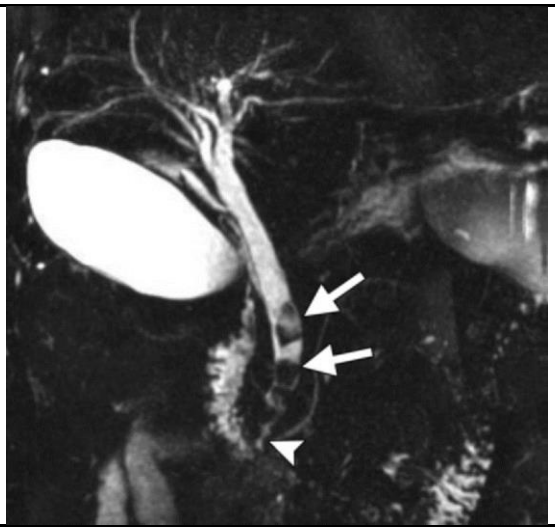
- Choledocholithiasis may occur in 8%–18% of patients with symptomatic gallstones.
- Chronic inflammation secondary to persistent biliary calculi may result in scarring and stricture formation.

MRCP:

- Bile duct calculi = multiple filling defects with angular margins
- CBD stricture

MRI:

- Minimal wall thickening and enhancement of the narrowed segment



Pancreas

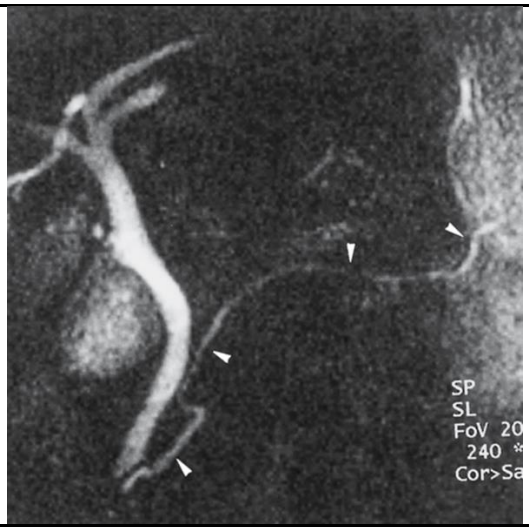
❖ List of Contents:

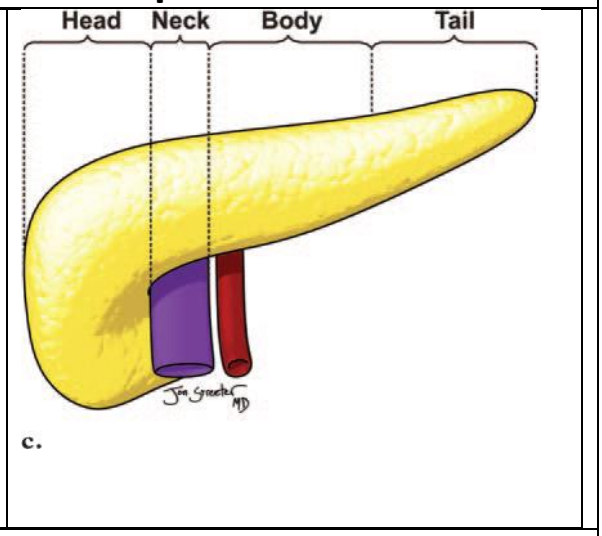
- **Anatomy**
- **Imaging Techniques**
- **Pancreatitis**
- **Focal Cystic Lesions**
- **Focal Solid Lesions**
- **Trauma**

Pancreatic Anatomy

- The pancreas is a coarsely lobulated gland
- Site: Anterior pararenal space of the retroperitoneum.
- Size:
  - Length = 15–20 cm
  - AP diameter of the head = 3.5 cm
  - AP diameter of the body = 2.2 cm

❖ Pancreas can be divided into four parts:





Pancreatic head:

- Located within the curve of the duodenum
- Right of SMV

Uncinate process

- is prolongation of caudal part of the head, which is oriented toward left.

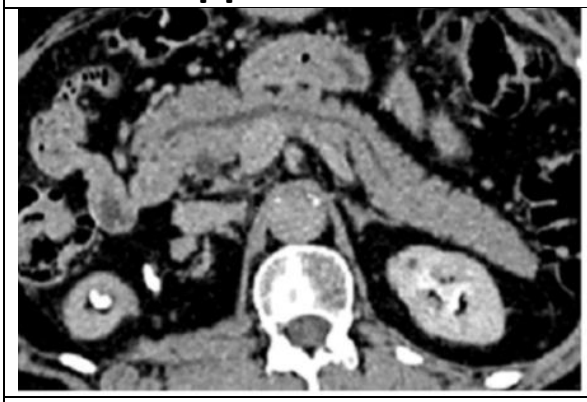
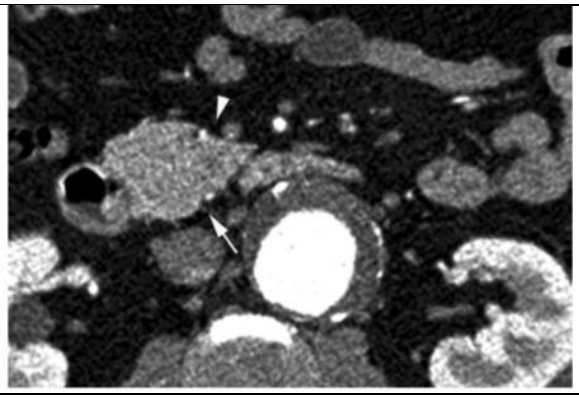
Pancreatic neck

- Ventral to SMV.

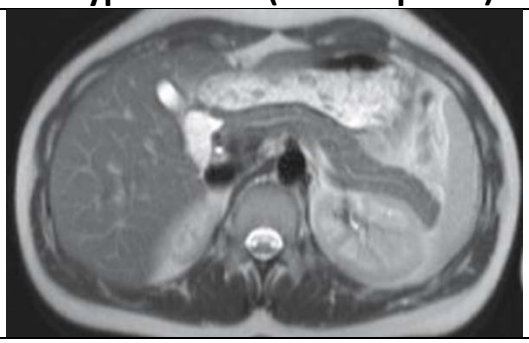
Pancreatic body and tail

- Behind the lesser sac and the stomach.
- The border between the body and tail is not clearly defined but can be determined using one-half of the distance between the neck and the end of the pancreas

❖ CT appearance of Normal Pancreas:

❖ MRI appearance of Normal Pancreas:

T1: high intensity (> SI of liver)	**T2: Hypointense (< SI of spleen)**
	
T1 FS	T1 FS + C

❖Normal Main Pancreatic Duct

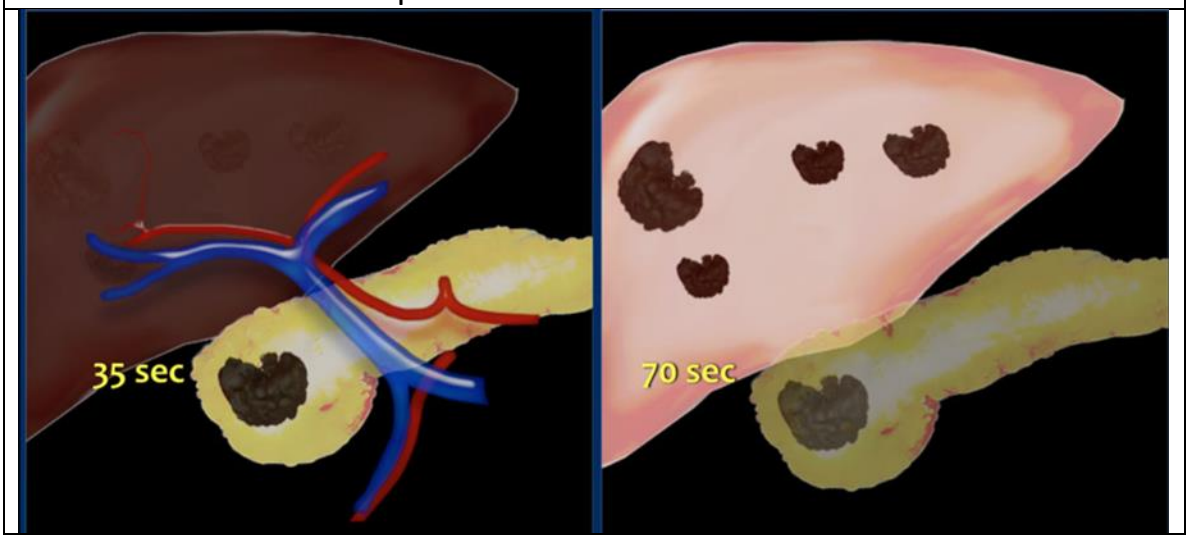
- Length of duct: Ranging from 9.5 to over 25 cm
- Size Reported maximum diameter:<5mm (increases with age)
- The caliber is greatest in the head and tapers smoothly to the tail
- Common types of course:
    - “Sigmoid” configuration: (ascending horizontal ascending; 34.5%)
    - “Pistol” configuration: (ascending horizontal horizontal; 27.6%)

Pancreas Imaging Modalities

→ **US**
→ **CT**
→ **MRI**
→ **Endoscopic US**
→ **Intraoperative US**

❖CT protocol of Pancreas:

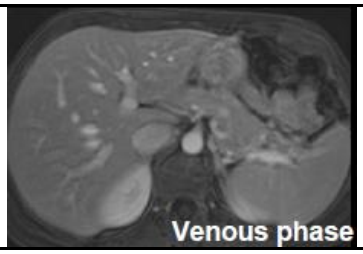
- **<u>NCCT</u>**
  - Detection of calcification
- **<u>Late arterial phase = Early Portal phase</u>**
  - 35-40 sec post contrast injection
  - Normal glandular tissue enhances optimally
  - Hypovascular tumor does not enhance
- **<u>Hepatic phase = Portal venous phase:</u>**
  - 70 – 80 sec post contrast injection
  - Detection of hepatic metastasis

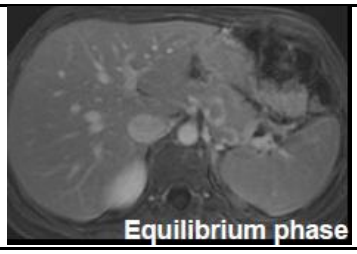


❖MRI Protocol of Pancreas:

- Axial T1 in-of-phase
- Axial T1 out-of-phase
- Axial T2 SSFSE (half single-shot fast spin-echo sequence)
- Axial T2 FS
- Coronal T2 SSFSE
- MRCP 2D slab
- MRCP 3D
- MRCP Secretin
- Axial DWI
- Axial ADC
- Axial T1 FS Dynamic:
    - Pre-contrast
    - Early arterial phase
    - Late arterial = Portal phase
    - Portal venous phase
    - Delayed phase = Equilibrium phase

- FOV = 360 mm
- Slice thickness = 3mm
- Slice gap = 0





❖MR Pancreatography with Secretin

- Improves visualization of pancreatic ductal system
- Improves the detection of:
    - Normal ducts, pancreatic divisum, ductal stricture
    - Chronic pancreatitis associated with marked ductal dilatation
- IV administration of 1 mL of secretin per 10 kg of body weight
- Image before and repeat every 2min for 16min after administering secretin
- Abnormal if duct remains greater than 1mm above baseline after 6min

Congenital Anomalies and Normal Variants of Pancreaticobiliary Tract and Pancreas

1) Main pancreatic duct:

Wirsung + Santoini ducts => fuse into common main pancreatic duct => drain via major duodenal papilla

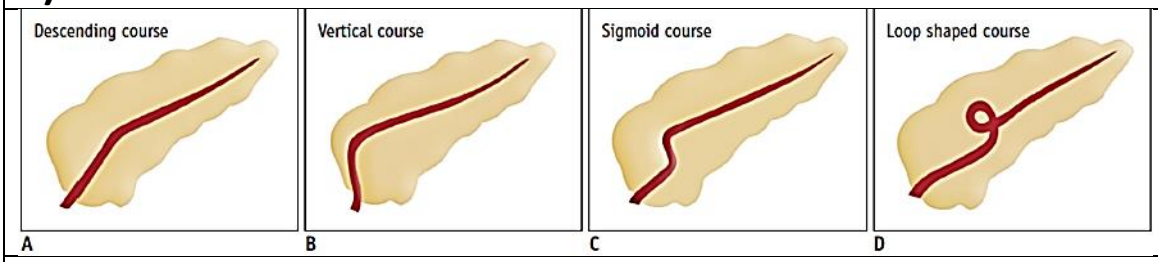
Duct of Wirsung:

Dorsal bud duct (part of the head and uncinate process)

Duct of Santorini:

Ventral bud duct (part of the head, body and tail of the pancreas).

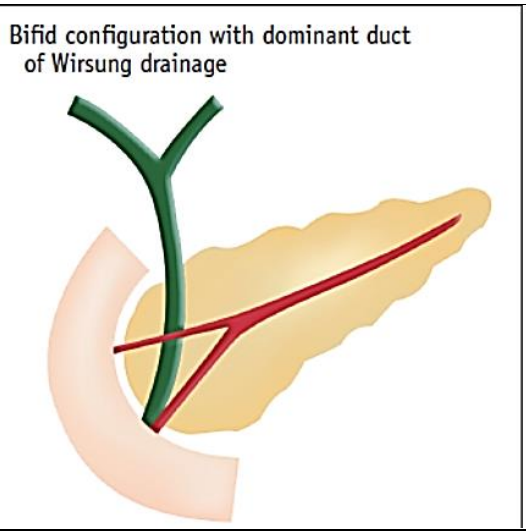
2) Variation In Course of Pancreatic Duct

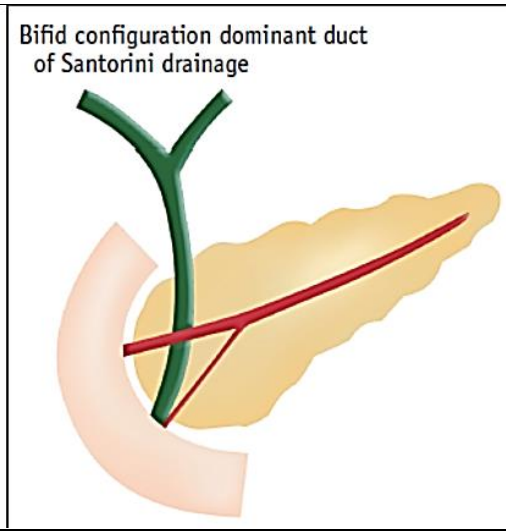


3) Bifid Configuration:

Duct of Santorini: Drains via minor papilla.

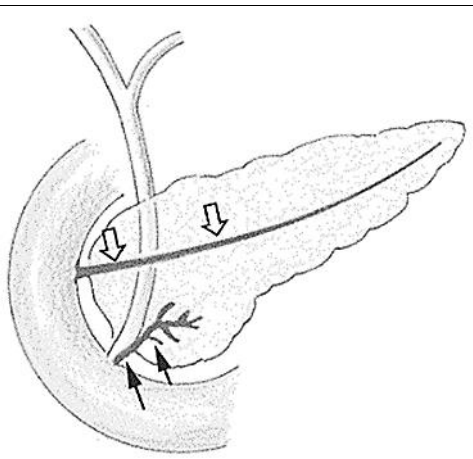
Duct of Wirsung: Drains via major papilla.

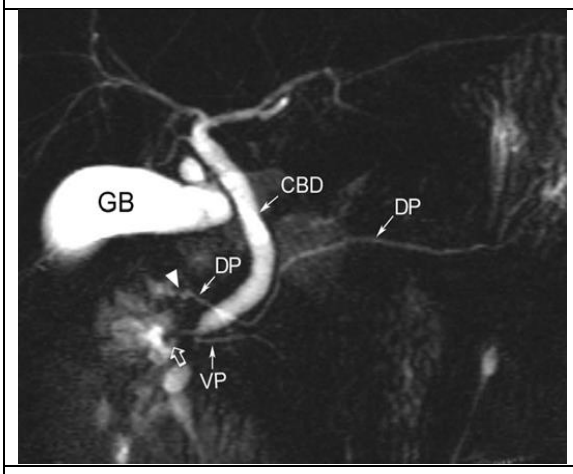




4) Pancreatic Divisum

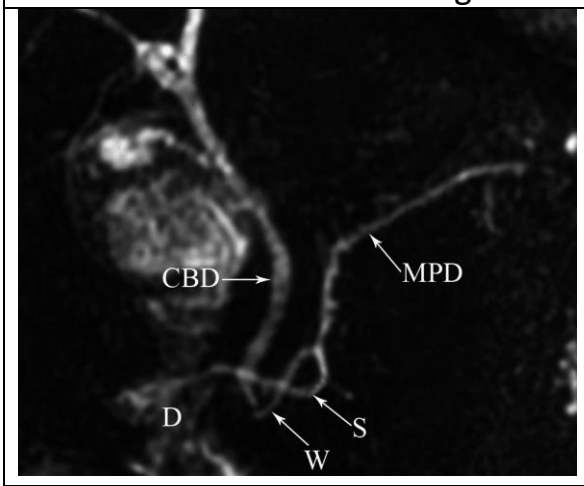
Small ventral duct (arrows)
Drains via major papilla.
Large dorsal duct (open arrows)
Drains via minor papilla.
Failure of fusion of dorsal and ventral pancreatic ducts

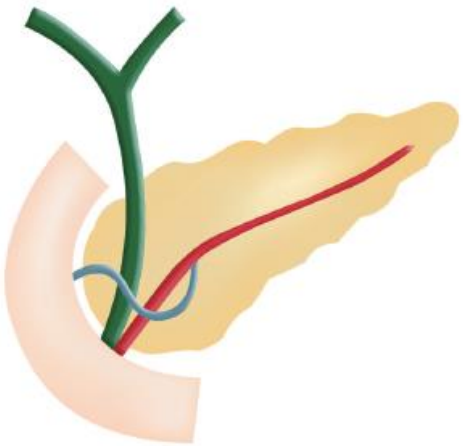




5) Ansa Pancreatica

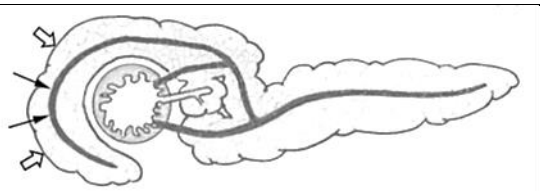
Duct of Santorini forms a reversed-S shape and connects with a side branch of the duct of Wirsung

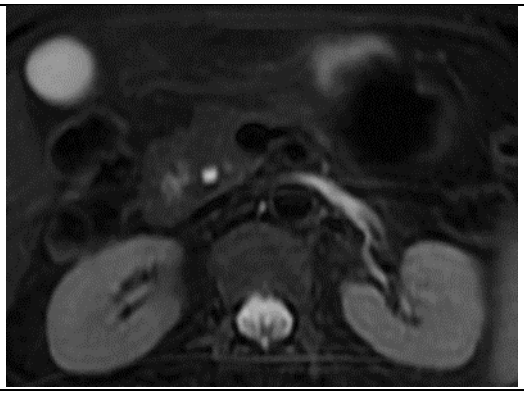




6) Annular Pancreas

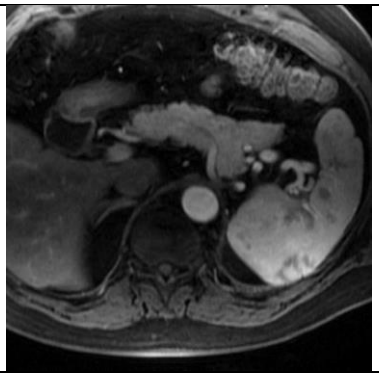
Pancreatic tissue surrounds the descending duodenum, either completely or incompletely, and is in continuity with the head of the pancreas





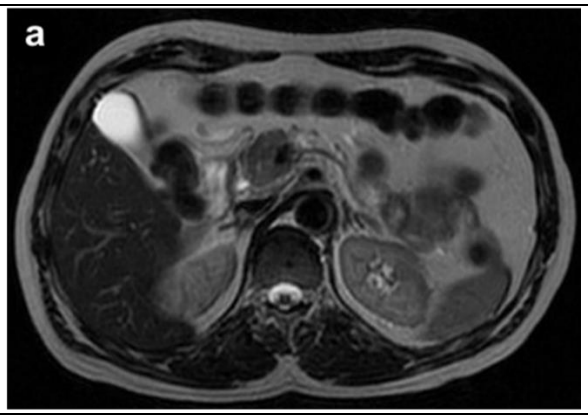
7) Bifid Pancreas

Rare branching anomaly of the pancreas tail and its duct system, also known as fishtail pancreas



8) Dorsal Pancreatic Agenesis

The pancreatic body and tail are hypoplastic or absent to varying degrees with preservation of the pancreatic head



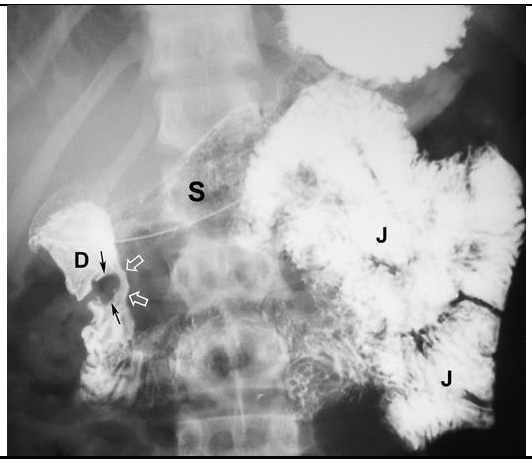
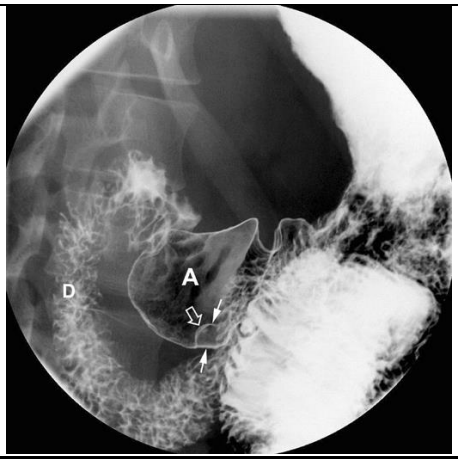
9) Ectopic Pancreatic Tissue (Heterotopia)

Pancreatic rests that lie outside and separate to the pancreatic gland

- Gastric antrum
- Proximal duodenum
- Ileum
- Meckel's diverticulae

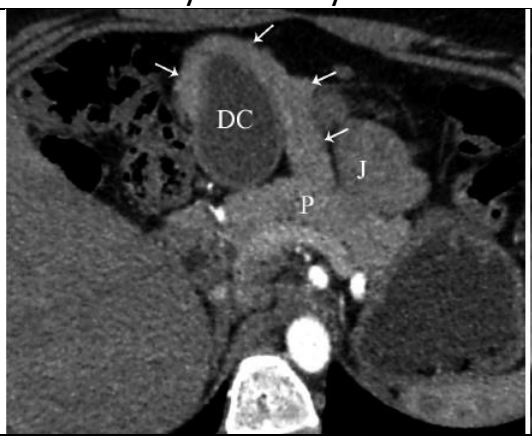
Imaging findings:

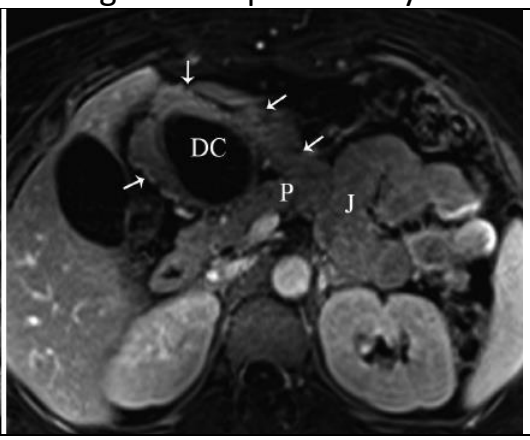
- Mural/submucosal filling defect
- Smooth overlying mucosa
- Central umbilication

Duodenum	Gastric antrum
	

10) Accessory Pancreatic Lobe

- An extremely rare anomaly,
- An accessory lobe of pancreatic tissue originating from the main pancreatic gland and containingan aberrant duct.
- This anomaly is usually associated with a gastric duplication cyst.



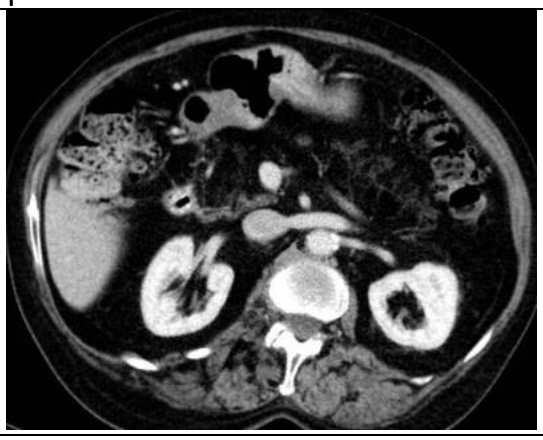
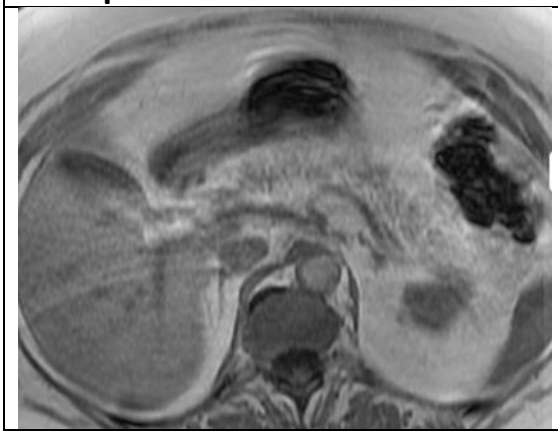
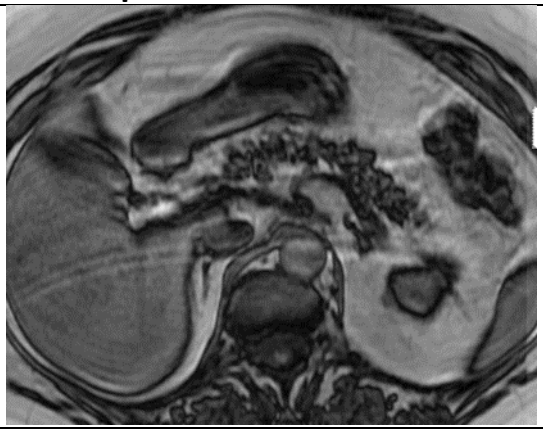


11) Fatty Replacement of Pancreas = Pancreatic Lipomatosis

- Fatty replacement of pancreatic parenchyma.

Aetiology:

- Obesity and aging
- Cystic fibrosis (most common cause in childhood)
- Metabolic/endocrine: hyperlipidemia, diabetes mellitus
- Storage disease: haemochromatosis
- Drugs: steroids
- Infectious: viral infection, sepsis

Pancreatic lipomatosis in young pateint with cystic fibrosis	Pancreatic lipomatosis in old, obese, diabetic, hyperlipidemic patient
	
In-of-phase	**Out-of-phase**
	

Acute Pancreatitis

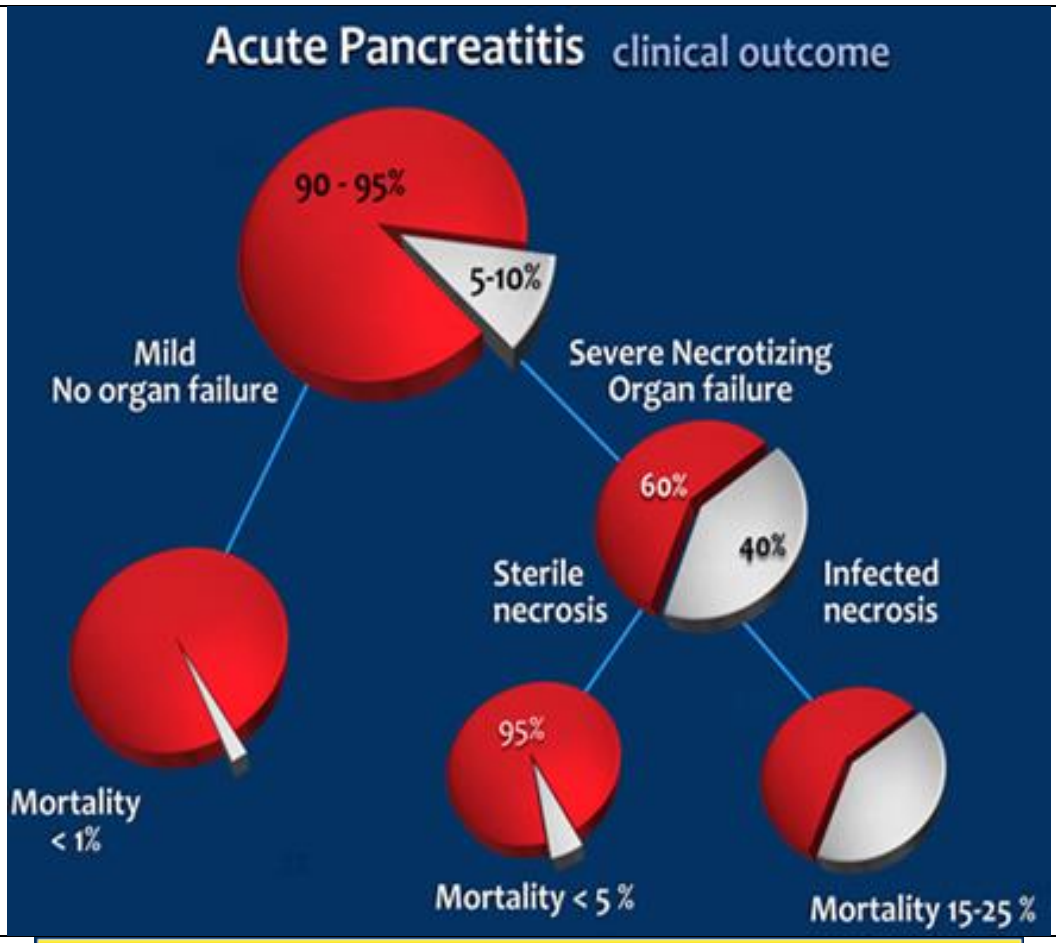
❖ Revised Atlanta Classification 2012

Diagnosis of Acute Pancreatitis => 3:

1) Abdominal pain:
   - Acute onset of persistent, severe, epigastric pain
   - often radiating to the back.
2) Serum lipase or amylase activity:
   - > 3 times greater than the upper limit of normal.
3) Characteristic imaging findings of acute pancreatitis:
   - Contrast-enhanced CT (CECT) and less commonly MRI or US.

Aetiology:

- Cholelithiasis (most common cause 80%)
- Alcohol consumption
- Idiopathic
- Trauma.
- Viral infection
- Systemic disease



CT Severity Index

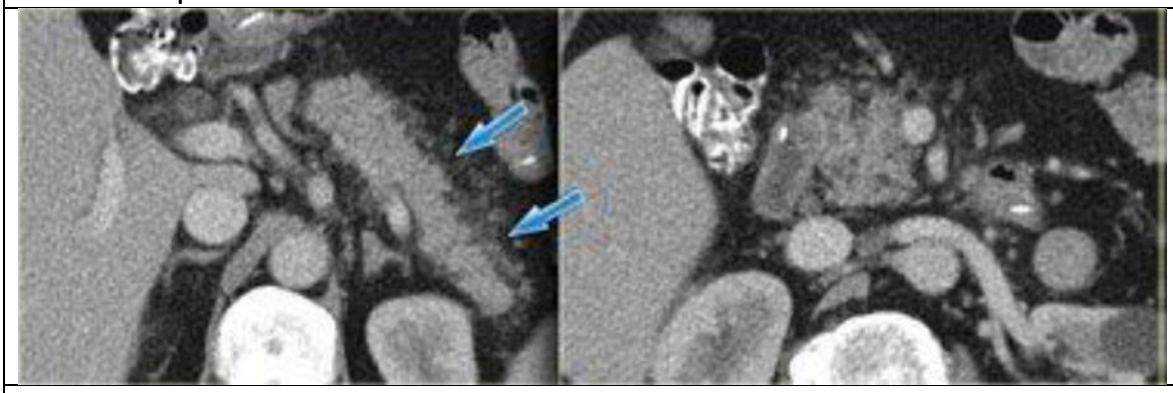
Pancreatic inflammation		**points**
Normal pancreas		0
Enlargement of the pancreas		1
Peripancreatic inflammation		2
1 acute peripancreatic fluid collection		3
⩾ 2 acute peripancreatic fluid collections		4
Pancreatic necrosis	None	0
	< 30%	2
	30% - 50%	4
	> 50%	6

Maximum 10 points

❖ 2 Types of Acute Pancreatitis

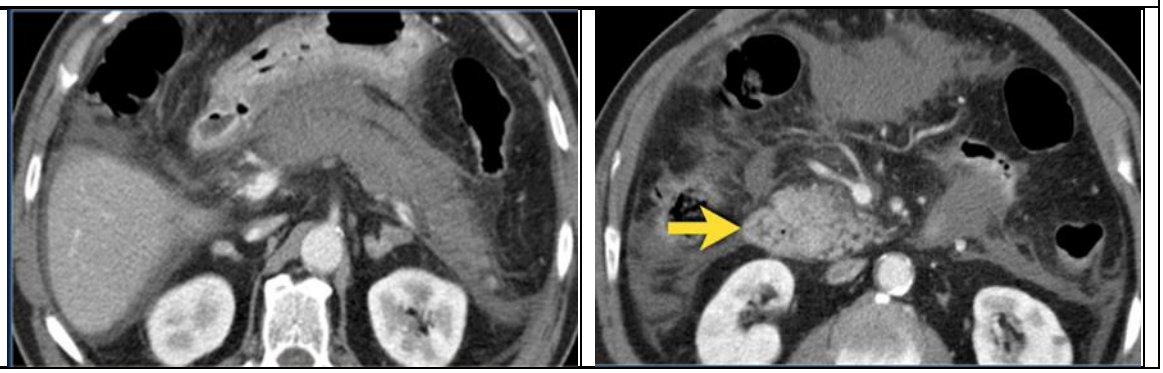
A- Interstitial Pancreatitis

- Focal or diffuse pancreatic enlargement
- Changes in density because of oedema
- Indistinctness of the margins of the gland owing to inflammation
- Surrounding retroperitoneal fat stranding
- Normal enhancement of the entire pancreatic gland
- No fluid collections
- No necrosis
- CTSI: 2 points.

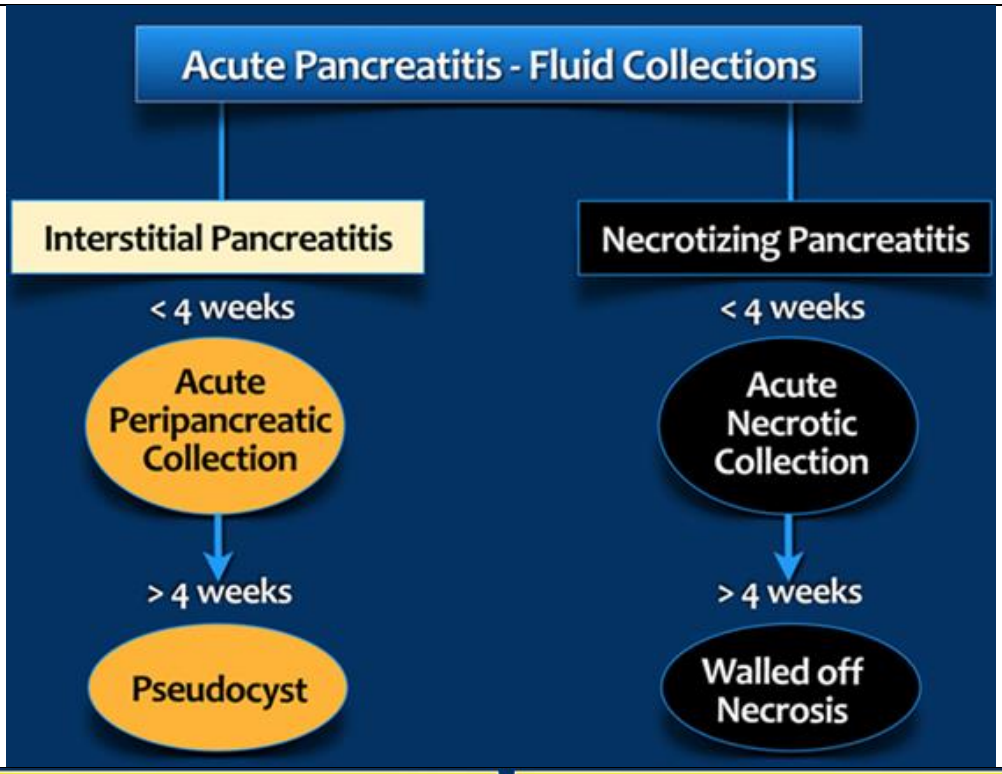


B- Necrotizing Pancreatitis

- Necrosis of pancreatic parenchyma or peripancreatic tissues
- 10-15 % of patients
- Lack of parenchymal enhancement



❖Peripancreatic Collections



Acute Peripancreatic Collection

- < 4 weeks
- In interstitial pancreatitis
- Homogeneous - fluid density
- *No fully definable wall*
- Adjacent to pancreas
- Confined by normal fascial planes

Acute Necrotic Collection

- < 4 weeks
- In necrotizing pancreatitis
- Heterogeneous collection
- *No fully definable wall*
- Intra- or extrapancreatic

Pseudocyst

- > 4 weeks
- In interstitial pancreatitis
- Homogeneous - fluid density
- *Well defined wall*
- Adjacent to pancreas
- No non-liquid component

Walled-off Necrosis

- > 4 weeks
- In necrotizing pancreatitis
- Heterogeneous collection
- *Well-defined wall*
- Intra- or extrapancreatic

1) Acute Peripancreatic Fluid Collection - APFC

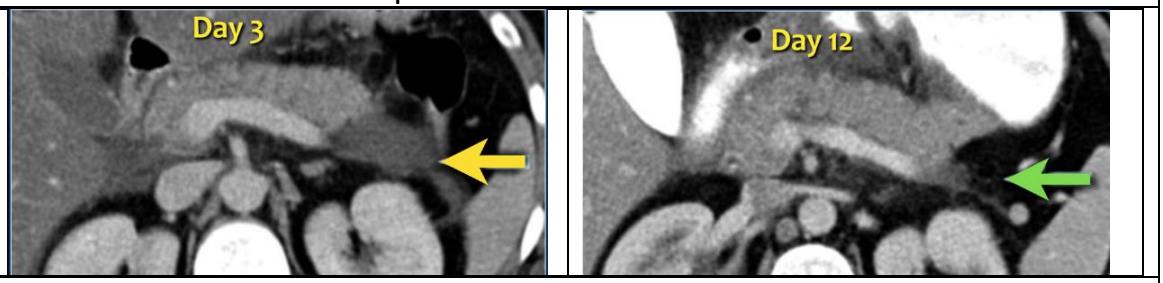
- In interstitial pancreatitis < 4w
- Intraabdominal fluid collections.
- No wall or capsule.

<u>Preferred locations of fluid collections are:</u>

- Lesser sac
- Anterior and posterior pararenal space of the retroperitoneum.
- Transverse mesocolon
- Small bowel mesentery

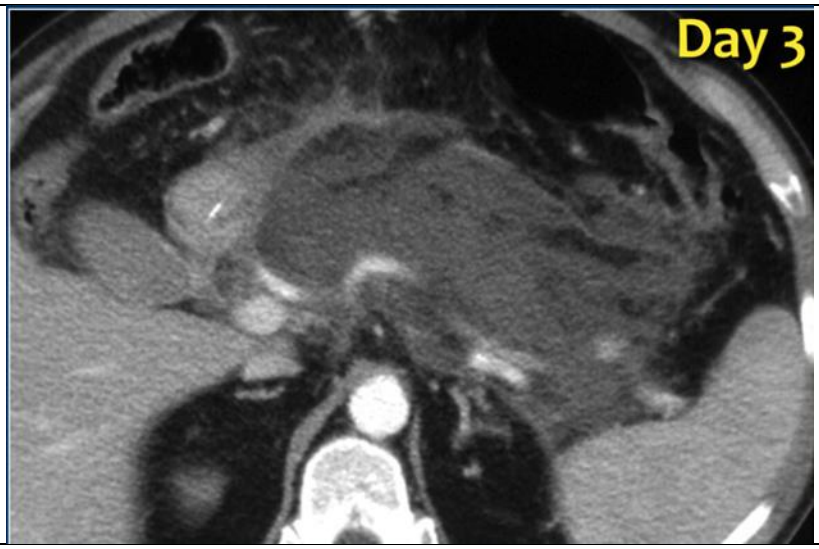
⇨ 50% of these collections show spontaneous regression (figure).

⇨ 50% either remain stable or increase and undergo organization and demarcation with liquefaction.



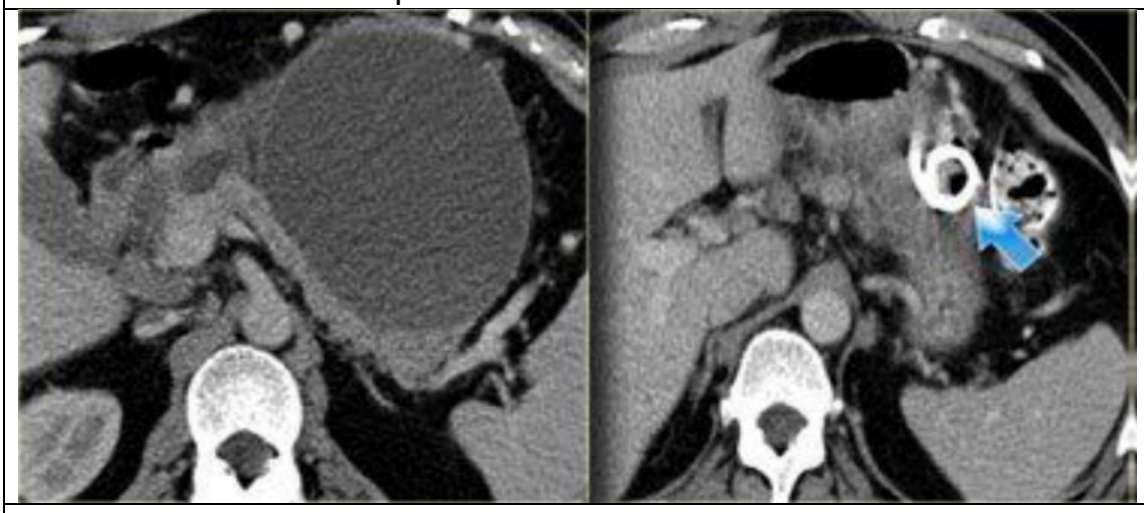
2) Acute Necrotic Collection - ANC

- Necrosis of the pancreas
- Inhomogeneous collection in the peripancreatic tissue
- No wall



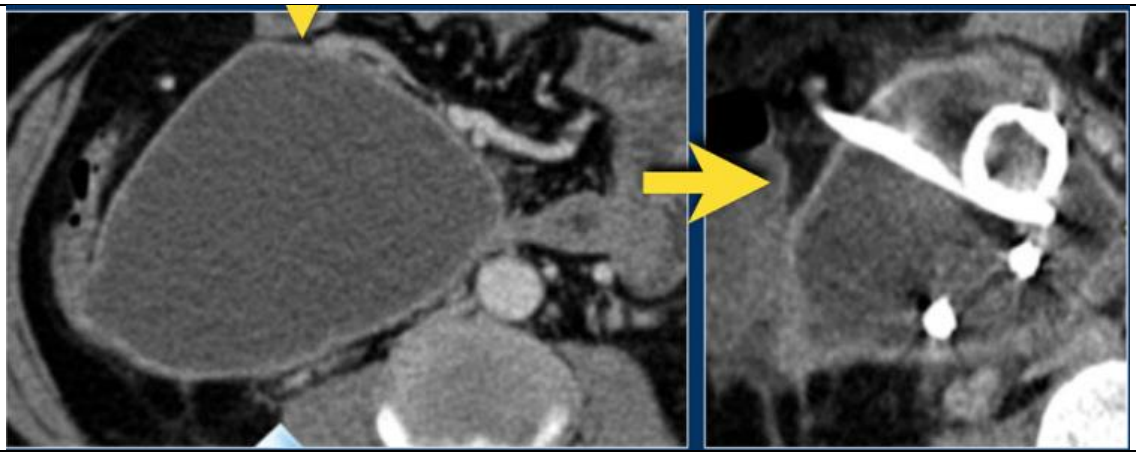
3) Pancreatic Pseudocyst

- It occurs in interstitial pancreatitis and the absence of necrotic tissue
- A well defined encapsulated pancreatic juice or fluid collection enclosed by a complete wall of fibrous tissue
- Requires at least 4 weeks to develop
- 50% will spontaneously resolve
- 50% require catheter or surgical drainage.
- True pseudocysts are uncommon, since most acute peripancreatic fluid collections resolve within 4 weeks.
- Most collections that persist after 4 weeks are walled-of-necrosis.



4) Walled-off Necrosis - WON

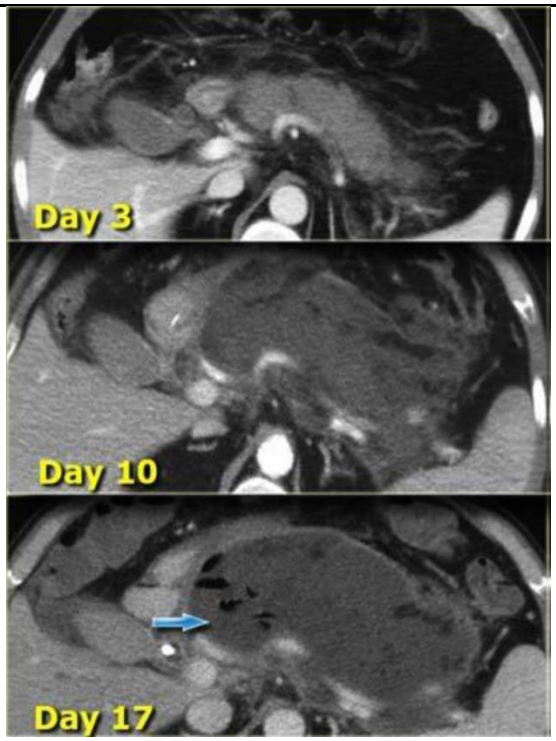
- CT can't differentiate between collection contains fluid only or a mixture of fluid and necrotic tissue
- C can't differentiate between infected or sterile collections.



❖ Complications:

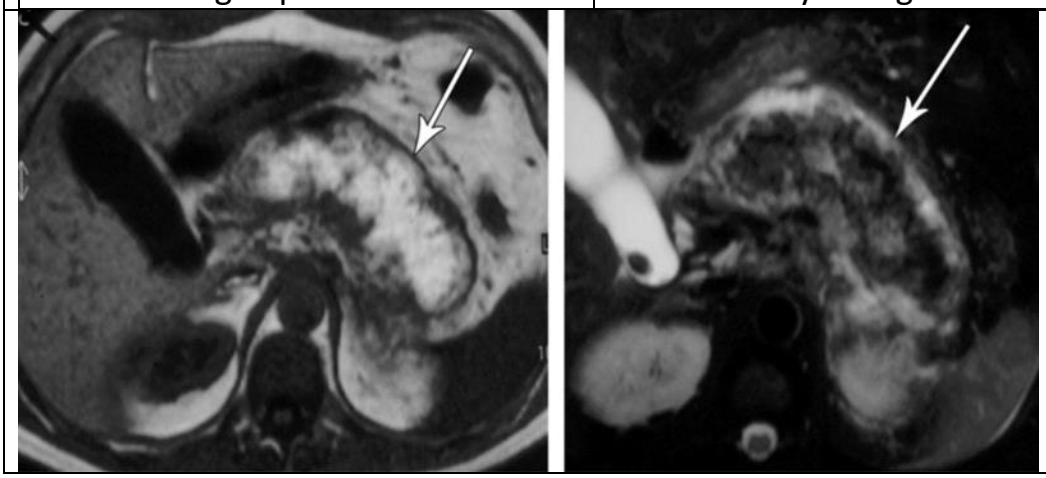
1) Infected necrosis

- Infection of necrotic pancreatic parenchyma or extrapancreatic fatty tissue - i.e. infected ANC or infected WON, depending on degree of encapsulation.
- Usually occurs in the 2nd-4th week and rarely in the first week.
- Most severe local complication of acute necrotizing pancreatitis.
- Most common cause of death in patients with acute pancreatitis.
- Diagnose infected necrosis when there are gas bubbles on CT (seen in 40%) or when FNA is positive for bacteria.



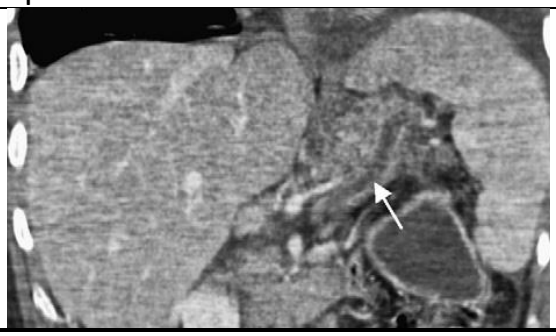
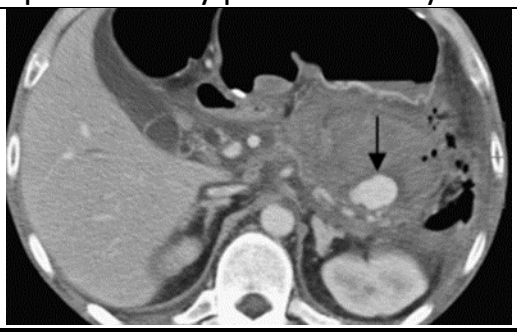
2) Hemorrhagic Pancreatitis

T1: Hyperintense Met HB signal within enlarged pancreas	T2 FS: Peripancreatic edema + Inflammatory changes



3) Vascular Complications:

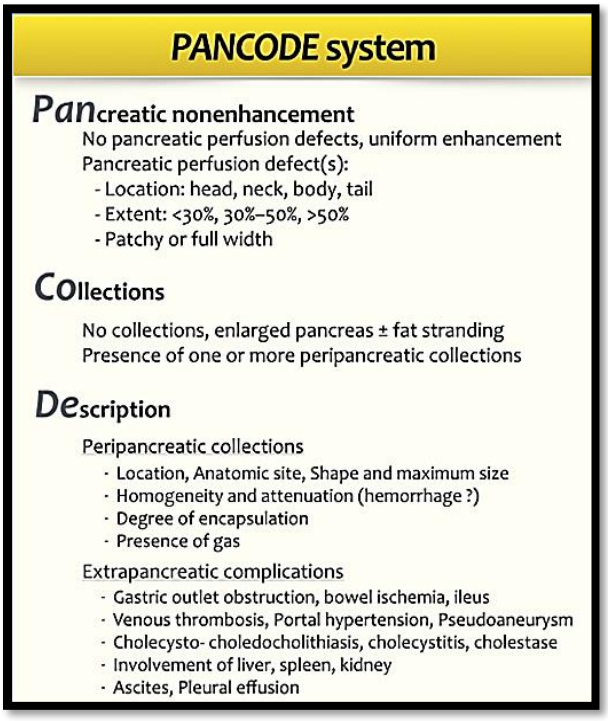
a) Hemorrhage: resulting from erosion of blood vessels and tissue necrosis
b) Splenic artery pseudoaneurysm: autodigestion of arterial walls by pancreatic enzymes results in pulsatile mass that is lined by fibrous tissue and maintains communication with parent artery
c) Splenic vein thrombosis
d) Portal vein thrombosis

Splenic vein thrombosis	Splenic artery pseudoaneurysm
	

4) Mass effect Complications:

a) Gastric outlet obstruction
b) Intestinal obstruction
c) Biliary obstruction

❖ Reporting - PANCODE


PANCODE system
Pancreatic nonenhancement
No pancreatic perfusion defects, uniform enhancement
Pancreatic perfusion defect(s):
- Location: head, neck, body, tail
- Extent: <30%, 30%–50%, >50%
- Patchy or full width
COllections
No collections, enlarged pancreas ± fat stranding
Presence of one or more peripancreatic collections
DEscription
Peripancreatic collections
- Location, Anatomic site, Shape and maximum size
- Homogeneity and attenuation (hemorrhage ?)
- Degree of encapsulation
- Presence of gas
Extrapancreatic complications
- Gastric outlet obstruction, bowel ischemia, ileus
- Venous thrombosis, Portal hypertension, Pseudoaneurysm
- Cholecysto- choledocholithiasis, cholecystitis, cholestase
- Involvement of liver, spleen, kidney
- Ascites, Pleural effusion

❖Management of Acute Pancreatitis

Current Management in Acute Pancreatitis

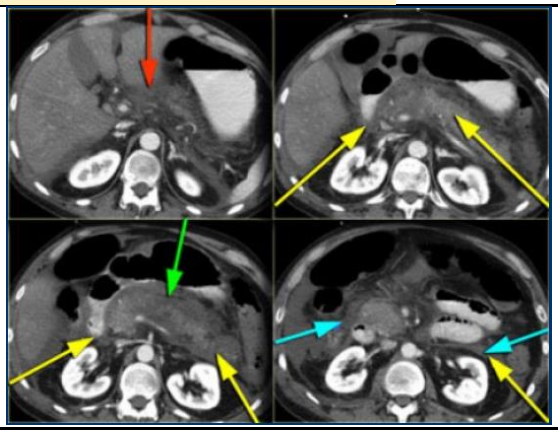
- Conservative approach
- Adequate pain relief
- Intravenous fluids and early enteral feeding
- No prophylactic antibiotics
- In case of multi organ failure ⟶ ICU

Radiological Intervention in Acute Pancreatitis

- FNA to differentiate between sterile and infected.
- Percutaneous drainage:
  - in case of pus under pressure
  - therapeutic drainage
  - when surgical intervention has to be postponed
  - as a guide for VARD-procedure

Percutaneous drainage of peripancreatic collections can be approached through:

⇨ Transhepatic (red arrow)
⇨ Transgastric (green arrow)
⇨ Transabdominal (blue arrows)
⇨ Retroperitoneal (yellow arrows)



Videoscopic assisted retroperitoneal debridement (VARD)

Chronic Pancreatitis

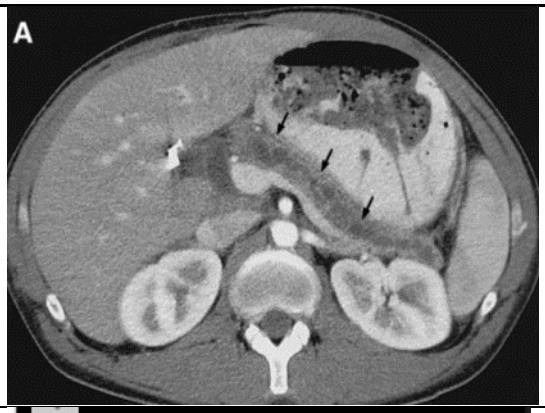
- represents the end result of a continuous, prolonged, inflammatory and fibrosing process that affects the pancreas.
- This results in irreversible morphologic changes and permanent endocrine and exocrine pancreatic dysfunction.

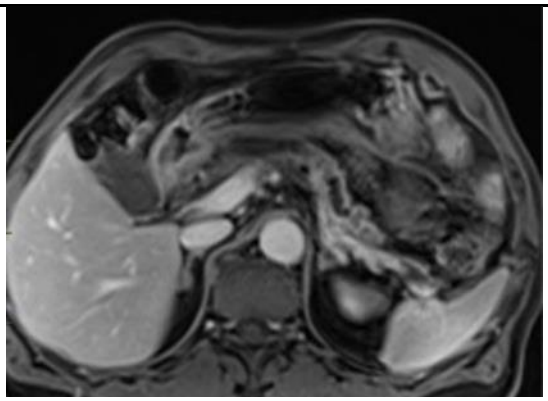
The most common cause of chronic pancreatitis in adults:

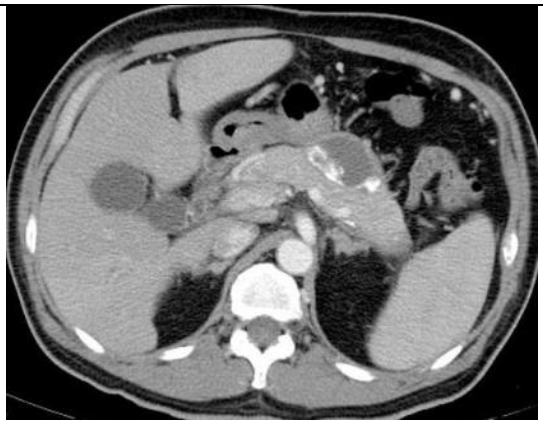
- Excessive alcohol consumption in developed countries
- Malnutrition in developing countries.

Imaging findings:

- Dilatation of the main pancreatic duct
- Pancreatic calcification
- Changes in pancreatic size (i.e. Atrophy), shape, and contour
- Pancreatic pseudocysts







Pancreatic Cystic Lesions

True Pancreatic Cysts:

Associated with:

- Von Hippel-lindau Disease (VHL)
- Autosomal-dominant Polycystic Kidney Disease (ADPKD)
- Cystic Fibrosis

Acute Pancreatitis Associated Collections:

- Pancreatic pseudocyst
- Walled off necrosis

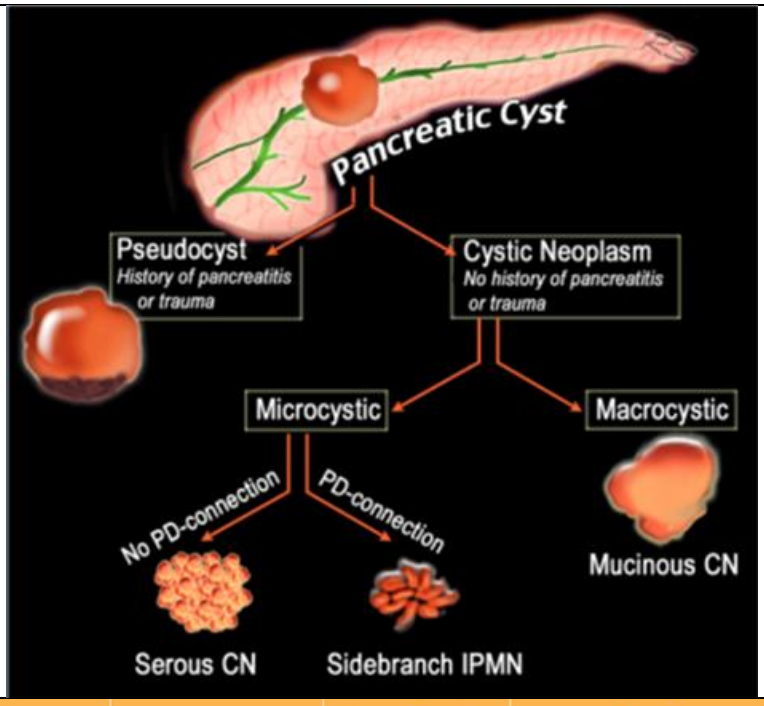
Primary Pancreatic Cystic Neoplasms

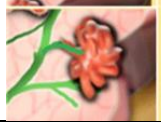
- Serous cystadenoma
- Mucin-containing cysts:
    - Mucinous nonneoplastic cyst
    - Mucinous cystadenoma
    - Mucinous cystadenocarcinoma
- Intraductal papillary mucinous neoplasms (IPMN)

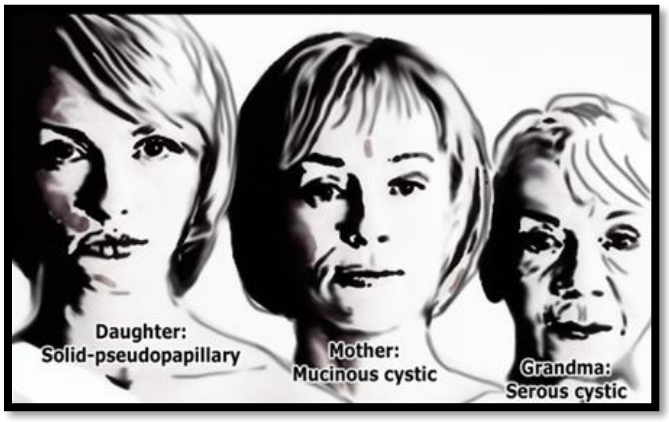
Primary Pancreatic Solid Neoplasms with Cystic Degeneration:

- Ductal adenocarcinoma with cystic features
- Solid Pseudopapillary Epithelial Neoplasm (SPEN)
- Cystic neuroendocrine tumors.

❖ Systematic Approach



		Age - Gender	Imaging
	SCN Benign	75% women 60-70 y **Grandma**	Lobulated microcystic 18% central scar with Ca^{++}
	MCN Malignant potential	99% women 40-50 y **Mother**	Macrocystic Usually 1 cyst 25% peripheral Ca^{++} 95% in tail and body
	Main-duct IPMN Malignant potential	M=W 60-80 y	Dilated Pancreatic duct Protruding papil of Vater
	**Side-branch IPMN** Malignant potential	M=W 60-80 y	Bunch of grapes connection to PD

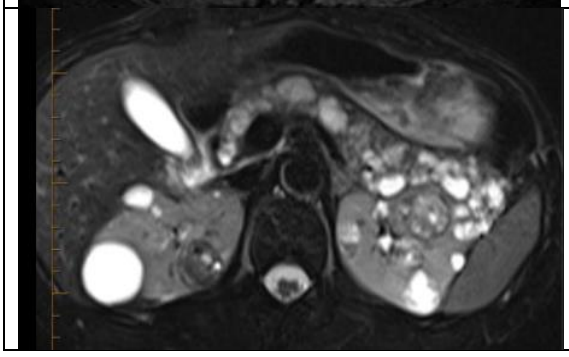
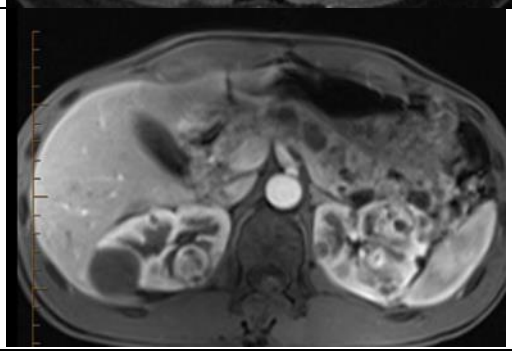


1) True Pancreatic Cyst

- Isolated true pancreatic cysts are rare
- Associated with:
  - Von Hippel-lindau Disease (VHL)
  - Autosomal-dominant Polycystic Kidney Disease (ADPKD)
  - Cystic Fibrosis

Imaging findings:

- Small (<1–2 cm in diameter).
- Unilocular cyst
- NO ductal communication
- NO perceptible wall
- NO internal septa.
- NO enhancement
- T1: hypointense
- T2: hyperintense

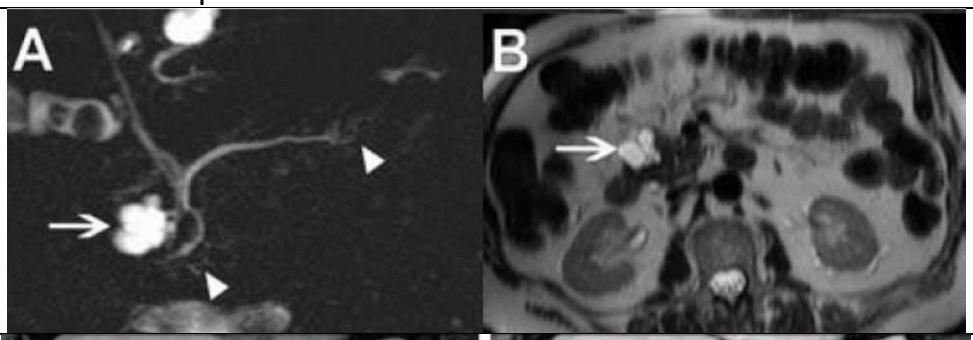
Axial T2 FS	Axial T1+C FS
	

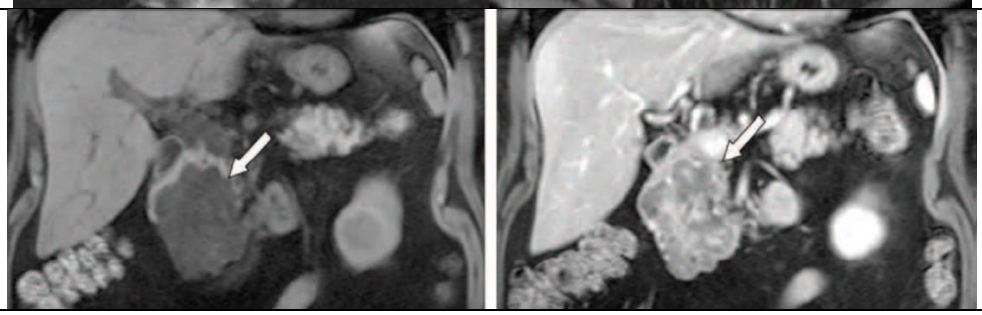
2) Serous Cystic Neoplasm

- Benign cystic neoplasms of the pancreas
- Sex: Women
- Age: > 60 y = Grandmother
- Multiple serous cystadenomas => Von Hippel–Lindau disease (VHL)

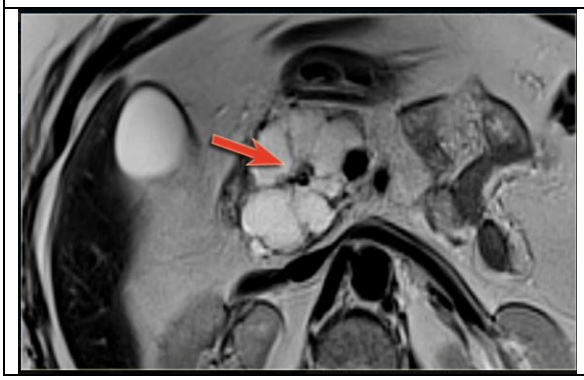
Imaging findings:

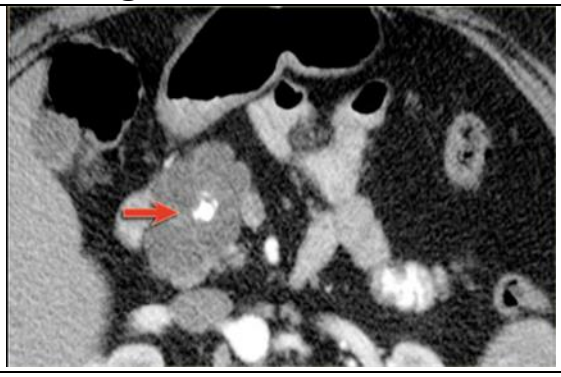
- Cluster of small cysts within the pancreas = Honeycomb like = grape like.
- No communication between the cysts and the pancreatic duct.
- T2: multilocular cysts show high signal intensity of simple fluid
- Central fibrous scar and may enhance on delayed phase images
- Central stellate pattern of calcification





Central scar with calcification: Low T2 signal



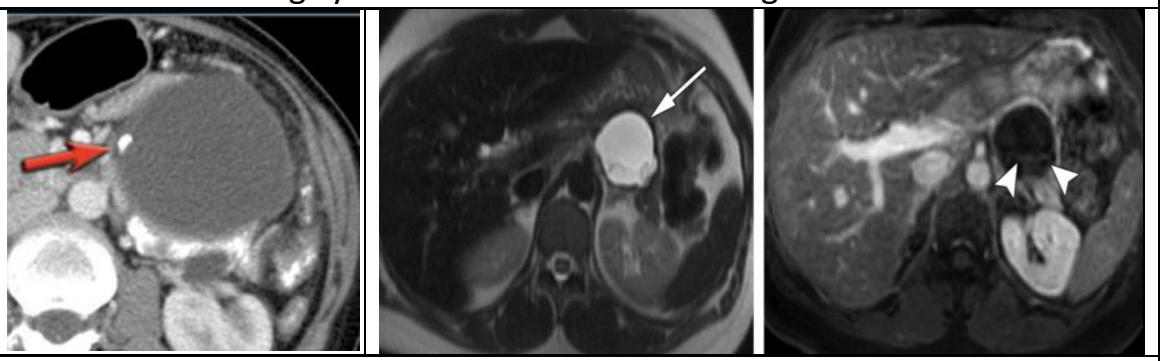


3) Mucinous Cystic Neoplasms

- 10% of pancreatic cystic neoplasms.
- Sex: >95% women
- Age: 40-60 years (Mothers)
- Location: Body or tail of the pancreas

Imaging Findings:

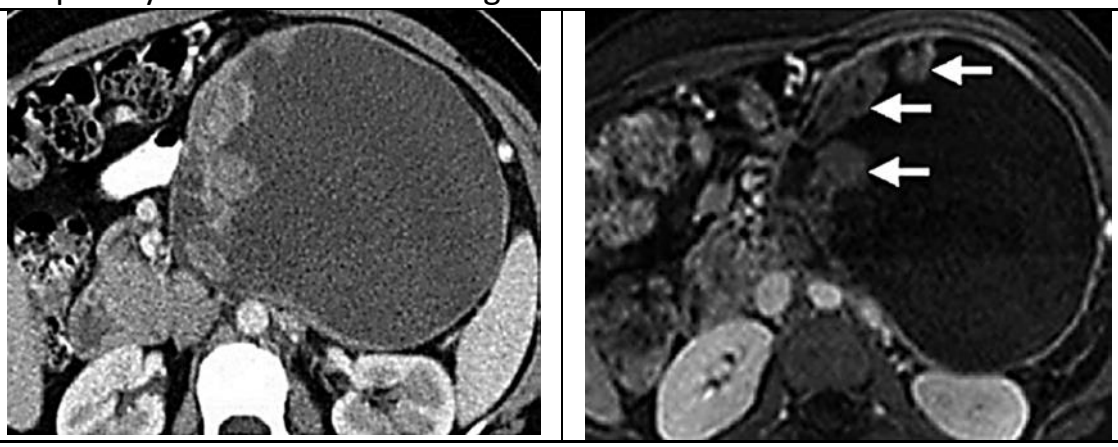
- Unilocular or mildly septate cystic lesion.
- No communication with pancreatic duct
- Thick wall / septations showing delayed contrast enhancement.
- Peripheral calcification (25%)
- Simple fluid containing cysts: homogeneous low T1 and high T2 signal
- Mucin-containing cysts: Increased intrinsic T1 signal.



- Because of their malignant potential, mucinous cystic neoplasms should be resected in all cases in which the patient is fit for surgery

Mucinous Cystadenocarcinoma

Complex cystic lesion + enhancing mural soft-tissue nodules



	Serous	Mucinous
Age	>60y	40-60y
Sex	F > M	
Location	Head	Body/Tail
No	>6	<6
Size	<20mm	>20mm
Enh	Hypervascular	Hypovascular
Ch	Central scar	--
Ca	Amarphous	Peripheral

4) IPMN = Intraductal Papillary Mucinous Neoplasm

- 20% of cystic lesions of the pancreas
- The tumors are characterized by:
- Intraductal papillary growth + abundant mucin production => Duct dilatation.
- Sex: Men
- Age: mean age, 65 years
- Location: head of the pancreas (75%)

Classification according to site of origin:

A. Main-duct IPMN: diffuse or segmental
B. Side-branch IPMN
C. Mixed IPMN: involving both the main duct and side branches

WHO => 3 subgroups:

(A) Benign neoplasms without dysplasia
(b) Borderline neoplasms (mild to moderate dysplasia)
(C) Carcinoma (either noninvasive or invasive)

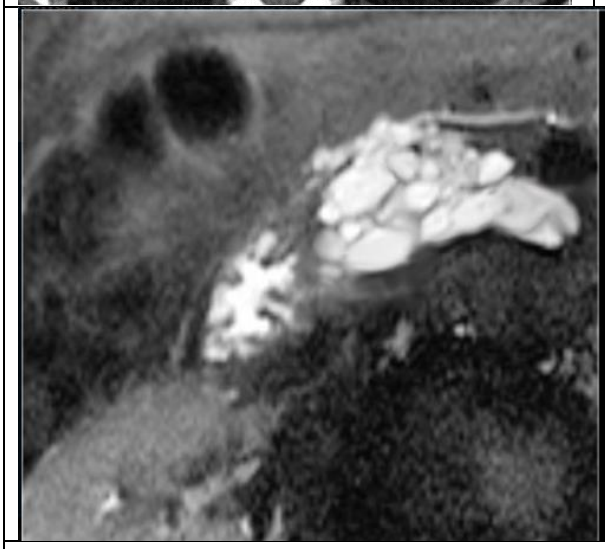
- 70% of main duct IPMNs
- 25% of side branch IPMNs

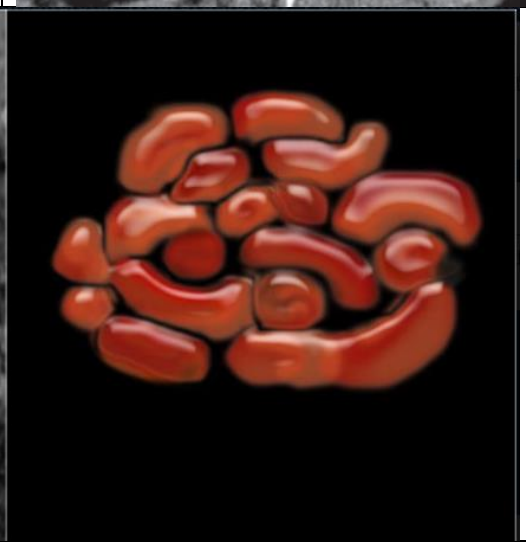
A- Main-duct IPMN:

- Diffuse or segmental dilatation of the main pancreatic duct.
- Hypointense T1 and hyperintense T2 signal

B-Side-branch IPMNs

i) Macrocystic pattern:	ii) Microcystic pattern:
Unilocular or multilocular cyst with few septa	Multilocular cyst with multiple thin septa



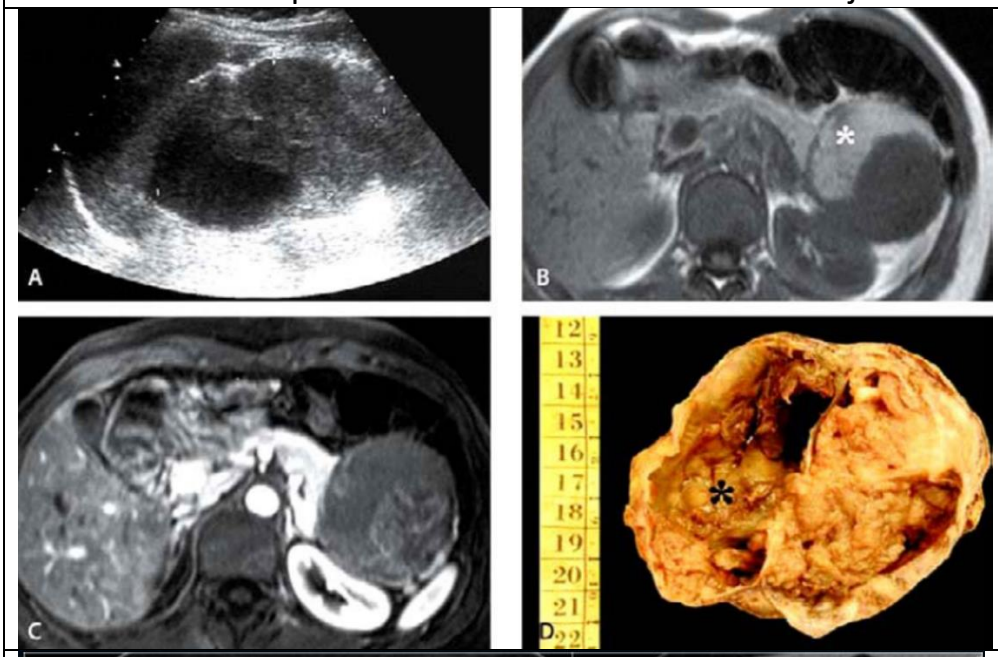


Malignant degeneration within main-duct IPMN

- Mural nodules,
- Focal solid components,
- Large unilocular cystic components,
- Enhancement of the main pancreatic duct wall,
- Main pancreatic duct diameter >18 mm.

5) Solid Pseudopapillary Tumors = Solid and Papillary Epithelial Neoplasm (SAPEN)

- 1% of all pancreatic Neoplasms
- Sex: predominantly in women
- Age: 20 – 30 y (Daughter)
- Low-grade malignant potential
- Good prognosis
- Site: Pancreatic tail.
- Internal hemorrhage, characterized by high T1 and low T2 signal
- A thick solid capsule that enhances after contrast injection.



Pancreatic Solid Lesions

- **Pancreatic Carcinoma:**
  - **Pancreatic Adenocarcinoma (85%)**
- **Solid Pseudopapillary Tumors (SAPEN)**
- **Pancreatoblastoma**
- **Pancreatic Lymphoma**
- **Pancreatic Metastasis**
- **Focal Pancreatitis**
- **Pancreatic Endocrine Tumors (PET) =**
  Islet Cell Tumors:
  - **Insulinoma**
  - **Gastrinoma**
  - **Glucagonoma**
  - **Vipoma**
  - **Somatostatinoma**
  - **Nonfunctioning**
  - **Poorly differentiated**

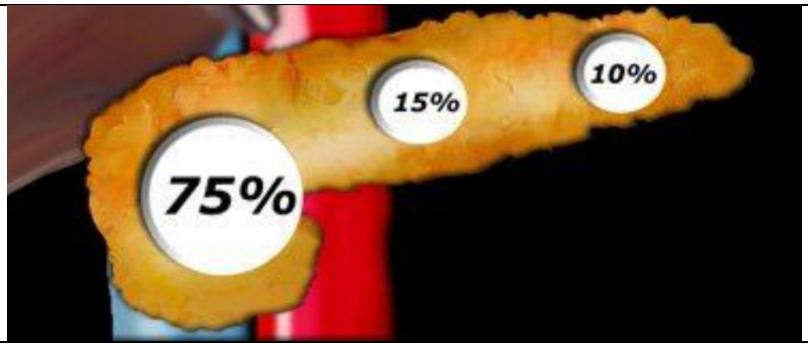
1) Pancreatic Carcinoma

- Pancreatic adenocarcinoma is most common malignant neoplasm of the pancreas (85%).
- Sex: Male > female
- Age > 60y in 80% of cases

Poor prognosis

- 80% unrespectable
- 40% Locally advanced tumor
- 40% Distant metastasis

❖Location:



Direct Sings:

- Ill defined lesion
- CT: hypodense
- T1 FS: hypointense relative to normal pancreas
- T2: hypointense relative to normal pancreas
- DWI: Restricted diffusion
- ADC: low

Indirect signs:

- Double duct sign:
  - Dilatation of intra and extrahepatic bile ducts and pancreatic duct
  - Abrupt termination of CBD and MPD at the level of the mass in the pancreas
  - Narrowing of the intrapancreatic portion of the CBD and MPD
- Bulky pancreatic head
- Discrepancy between size of pancreatic head, neck, body and tail
- Ratio between / transverse diameter of adjacent vertebra > 0.7

Dynamic CT or MRI Pancreatic Study:

- Arterial phase (20sec):
  - Assessment of SMA
- Pancreatic (40-50sec) phase:
  - The mass is hypodense / hypointense relative to normal pancreas; due to poor early enhancement
- Portal venous (70-90sec) and delayed (120sec) phases
  - The mass shows progressive enhancement over time on more delayed phases, due to the fibrotic nature of the mass.

Local Tumor Extension:

- Retroperitoneal extension:
  - Obliteration of the fat around the celiac axis or SMA
- Bowel (Duodenum and stomach):
  - Compression / invasion / obstruction / fistula
- Spleen:
  - Invasion

Vascular Involvement (SMA, SMV, PV, CHA, Celiac):

- Fat plane or normal pancreatic tissue between tumor and vessel
- Tumor / vessel interface ≤ 180
- Tumor / vessel interface > 180
- Focal vessel narrowing or contour irregularity (Tear drop / tethering)
- Venous thrombosis and collaterals

Regional LNs:

- Peripancreatic and porta-hepatis LNs

Distant Spread:

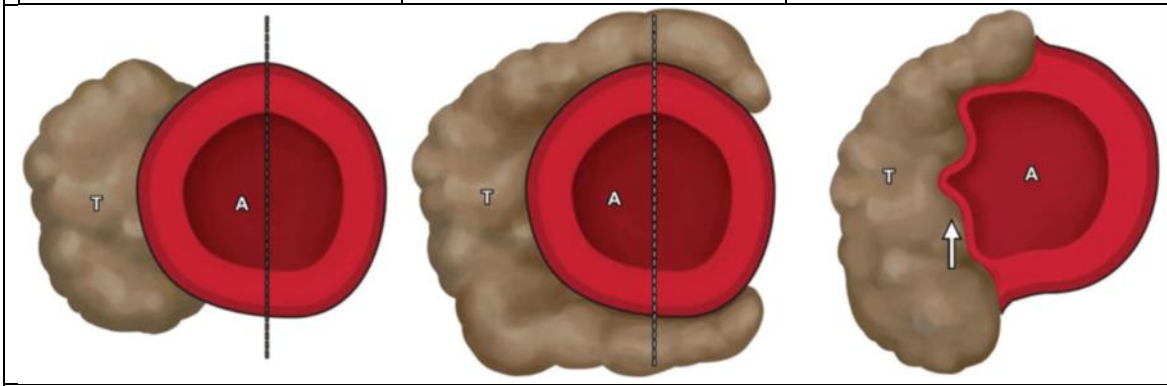
- Liver metastasis
- Peritoneal and omental nodules
- Non-regional LNs
- Ascites

Irresectability:

- Vessel tumor interface < 180 (Borderline respectable)
- Vessel tumor interface > 180 or occlusion
- Bowel invasion
- Distant metastasis

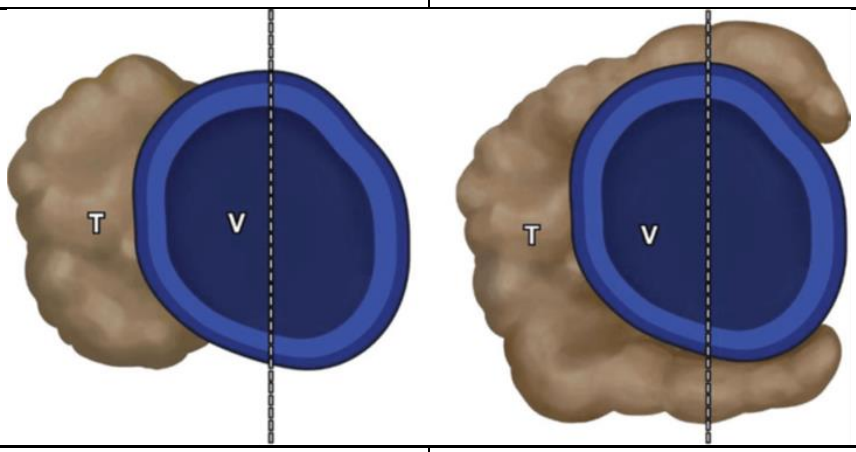
❖ Arterial Tumor Interface / Contact

< 180	> 180	Arterial Deformity

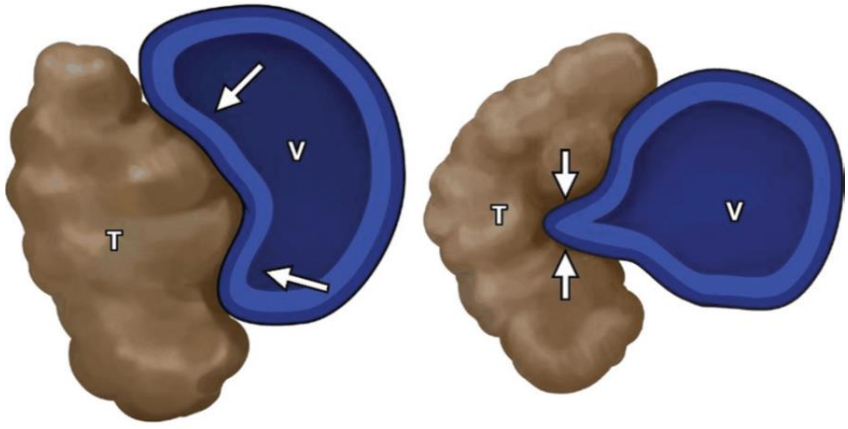


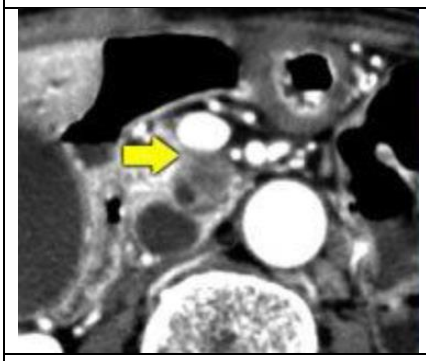
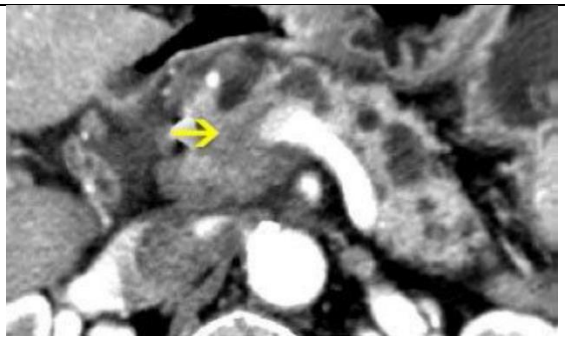
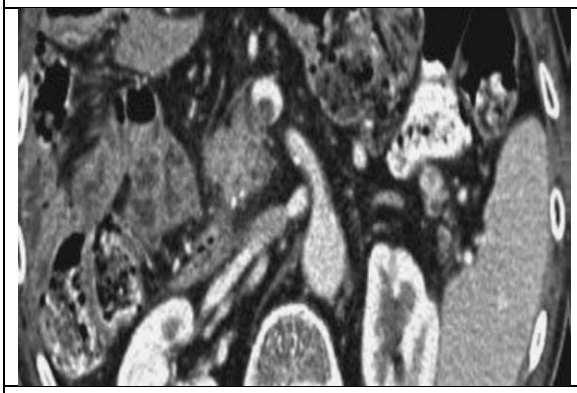
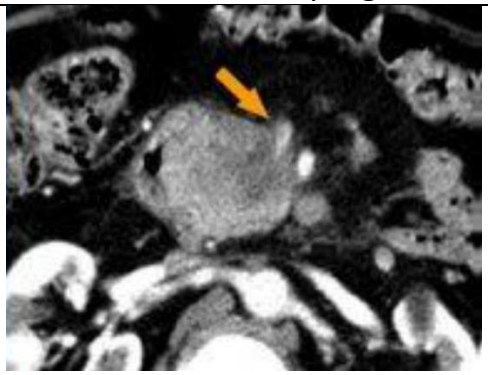
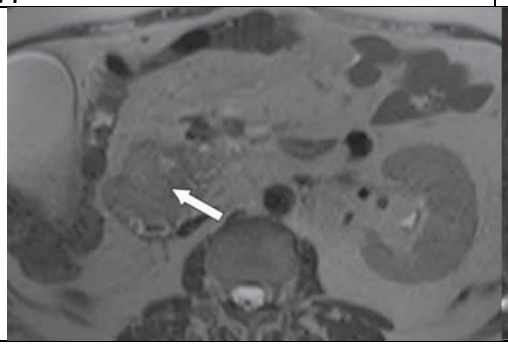
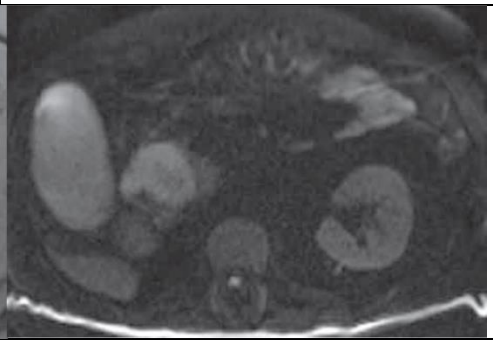
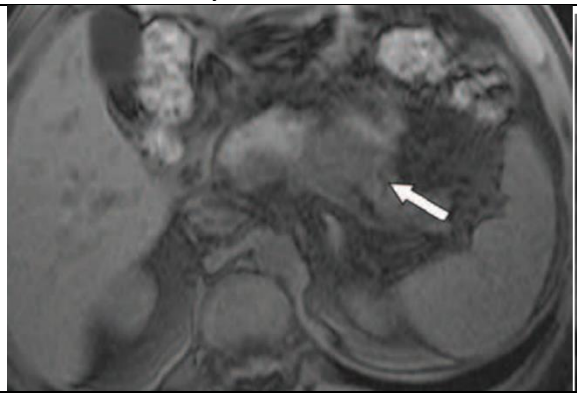
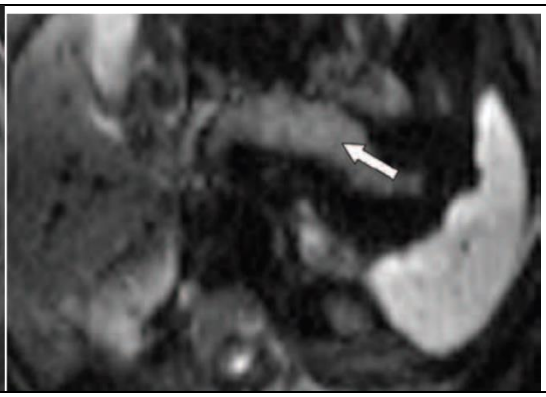
❖ Venous Tumor Interface / Contact

< 180	>180



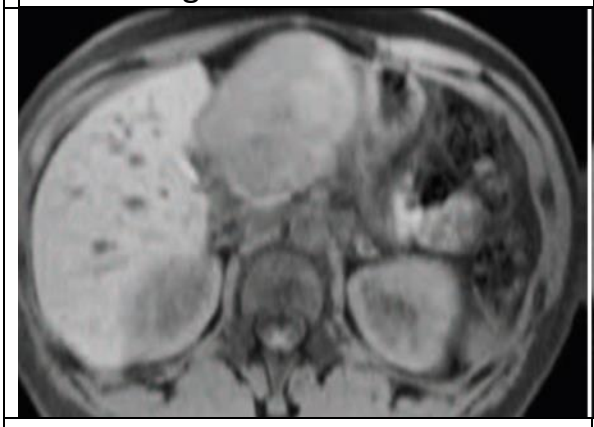
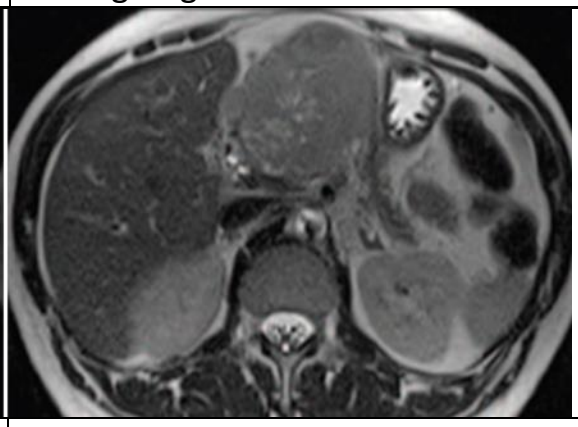
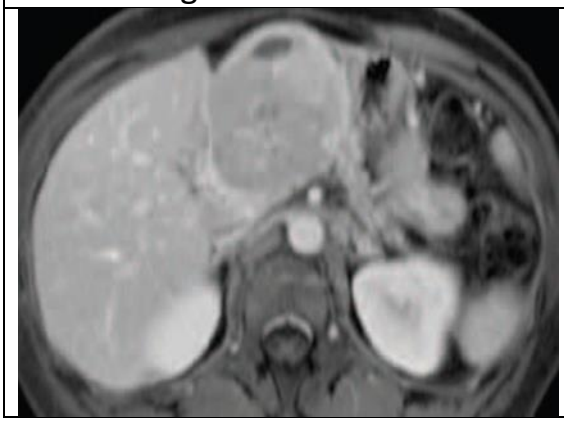
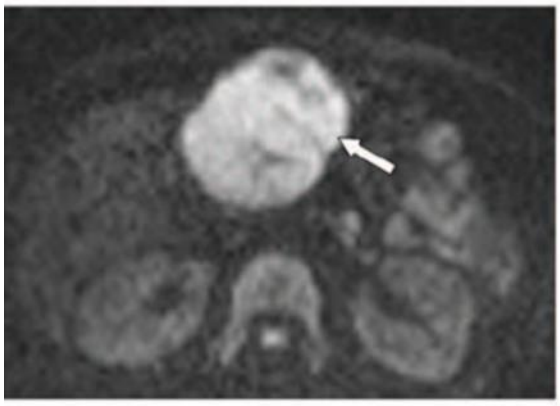
Venous Deformity	Tear drop deformity



Tumor-Vessel Interface <180°	Tumor-Vessel Interface >180°
	
SMV Tumoral thrombus	Mesenteric tear drop sign
	
Pancreatic head ill defined lesion	
T2: hypointesne	DWI: Restricted
	
Pancreatic body infiltrative lesion	
	

2) Pancreatoblastoma

- Rare tumor, < 1% of all pancreatic neoplasms
- Most common pancreatic tumor in children.
- Sex: prevalent in males.
- Age: bimodal distribution
    - 3–5 years
    - 25–30 years.

T1: Low signal	T2: High signal
	
+C: Heterogeneous enhancement	DWI: Restricted
	

3) Pancreatic Endocrine Tumors (PET)
Islet Cell Tumors

- 1%–2% of all pancreatic neoplasms
- Endocrine tumors are classified as functioning or nonfunctioning on the basis of the hormones that they produce .
- The dimensions can range from 0.5–1 cm to 10 cm.

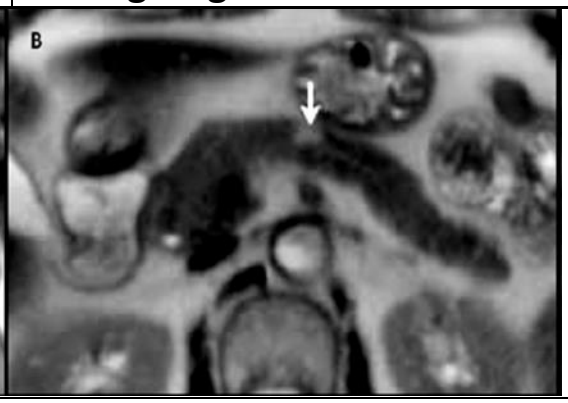
WHO Classification of PETs

Tumor Type	Benign or Malignant	Characteristics
Well-differentiated endocrine tumor	Benign	Confined to the pancreas, <2-cm diameter, no vascular or perineural invasion, less than 2 mitoses per 10 high-power fields, less than 2% Ki-67 positive cells
	Uncertain	Confined to the pancreas and at least one of the following: <2-cm diameter, vascular or perineural invasion, 2–10 mitoses per 10 high-power fields, greater than 2% Ki-67 positive cells
Well-differentiated endocrine carcinoma	Low-grade malignant	Gross local invasion or metastasis
Poorly differentiated endocrine carcinoma	High-grade malignant	>10 mitoses per 10 high-power fields

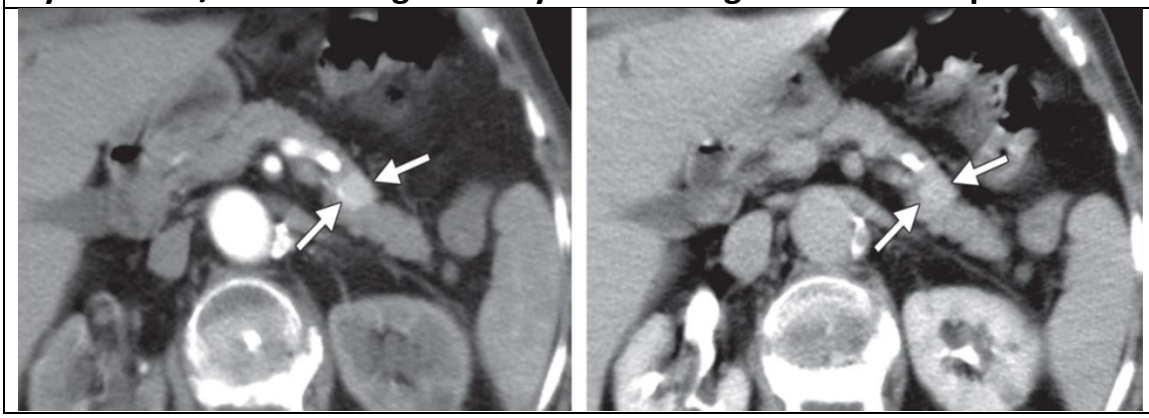
Type of PET	Associated Syndrome	Symptoms	Average Size (cm)	Most Common Location
Insulinoma	Whipple triad	Hypoglycemia, dizziness, vision changes, and palpitations	<2	Even distribution throughout the pancreas
Gastrinoma	Zollinger-Ellison syndrome	Peptic ulcer disease, diarrhea, and esophagitis	Duodenal, <1; pancreatic, 3–4	Duodenum, followed by the pancreatic head
Glucagonoma	4D syndrome	Necrolytic migratory erythema, diabetes, and thromboembolism	7–8	Pancreatic tail
Vipoma	WDHA syndrome	Profuse watery diarrhea, weight loss, and hypokalemia	5–6	Pancreatic tail
Somatostatinoma	Inhibitory syndrome	Diabetes, steatorrhea, diarrhea, and cholelithiasis	5–6	Pancreatic head
Nonfunctioning	None	Abdominal pain, mass, and weight loss; discovered incidentally	5–6	Even distribution throughout the pancreas
Poorly differentiated	None or paraneoplastic syndrome	Abdominal pain, cachexia, and jaundice	6	Pancreatic head

A) Insulinoma

- 1st common functioning PET
- 40% of all functioning PETs.
- Benign in the majority of cases.
- Size: 90%<2 cm in diameter and 40% <1 cm.
- **Location:** frequently in the body-tail of the pancreas.
- **Whipple triad:** hypoglycemia, low fasting glucose levels (< 50 mg/dl) and relief of symptoms with glucose administration.

T1: Low signal	T2: High signal
	

Dynamic CT/MRI: Homogenous hyerenhancing in the arterial phase

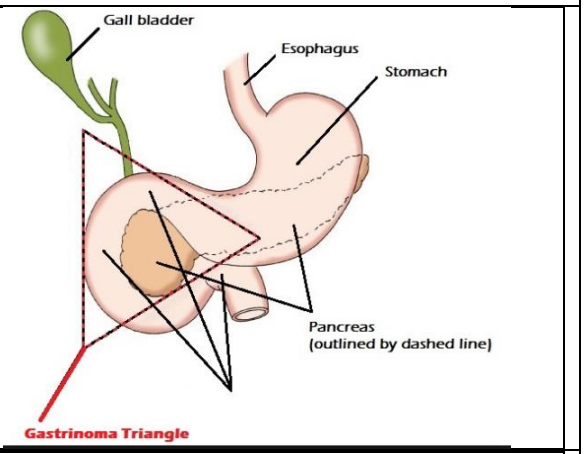


B) Gastrinoma:

- 2nd most common endocrine tumor.
- Unlike insulinoma, it is characterized by an elevated incidence of malignancy.
- **Zollinger-Ellison syndrome:** multiple gastric ulcers, thickened folds of stomach, duodenum & jejunum and diarrhea because of the excessive secretion of gastric acid.

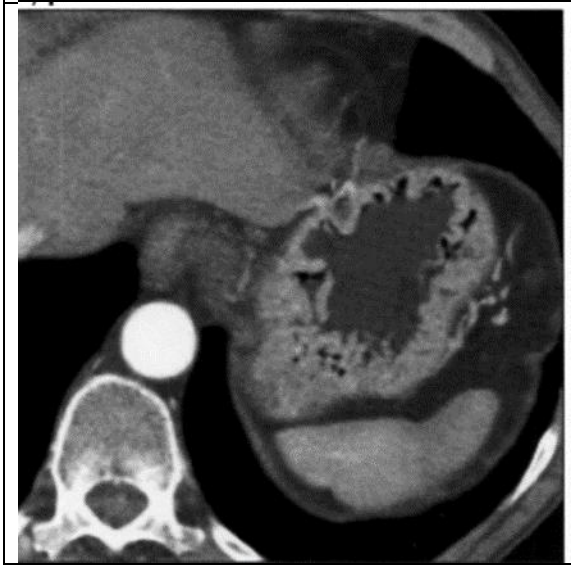
- **Multiple (60%)**
- **Malignant (60%)**
- **Metastases (30-50%)**

Location: Gastrinoma triangle, formed by the pancreatic head, the duodenum and the junction of the cystic and common bile duct.



Thickened gastric folds

Hypervascular pancreatic body head lesion



Periampullary Tumors

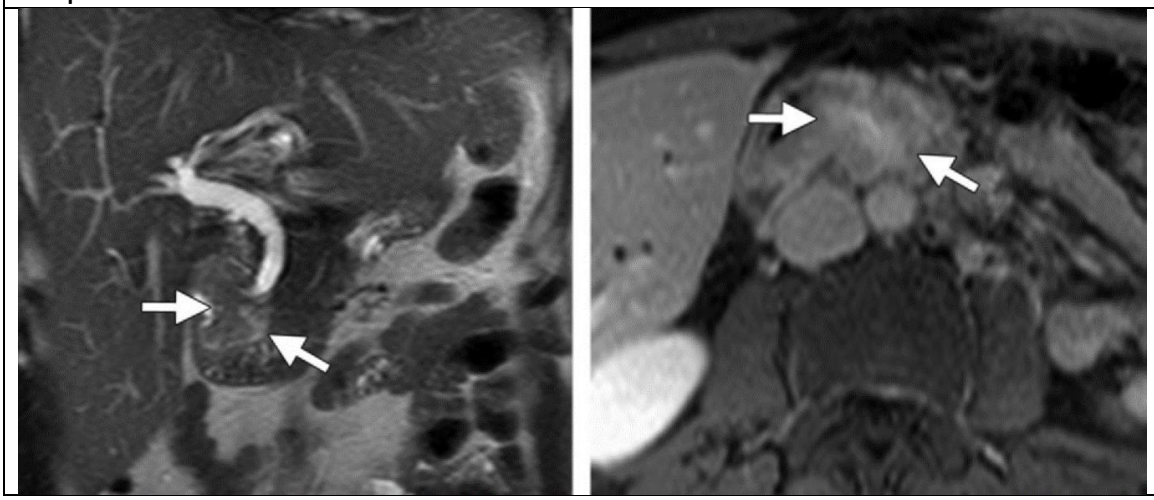
A. Ampullary tumors
B. Distal CBD tumors
C. Periampullary portion of duodenum
D. Head & uncinate process of pancreas.

A- Ampullary Tumors

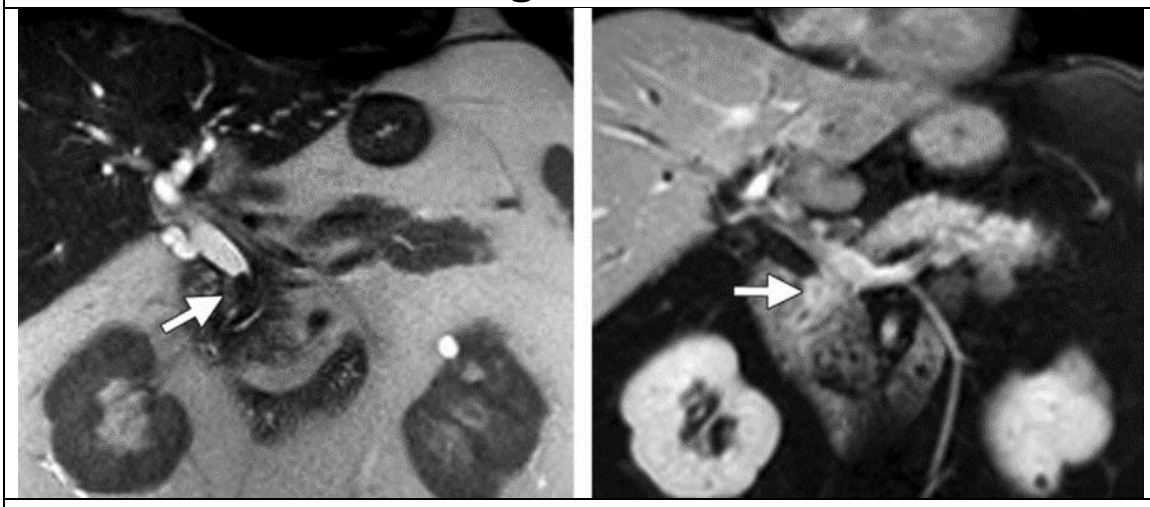
- The ampulla of Vater is located in the major duodenal papilla
- The ampulla of Vater represents the junction of the common bile duct (CBD) and main pancreatic duct of Wirsung
- The ampulla of Vater is surrounded by the sphincter of Oddi.
- Ampullary carcinoma is adenocarcinoma arising in the ampullary complex distal to the confluence of the pancreatic duct and CBD

Imaging finding:

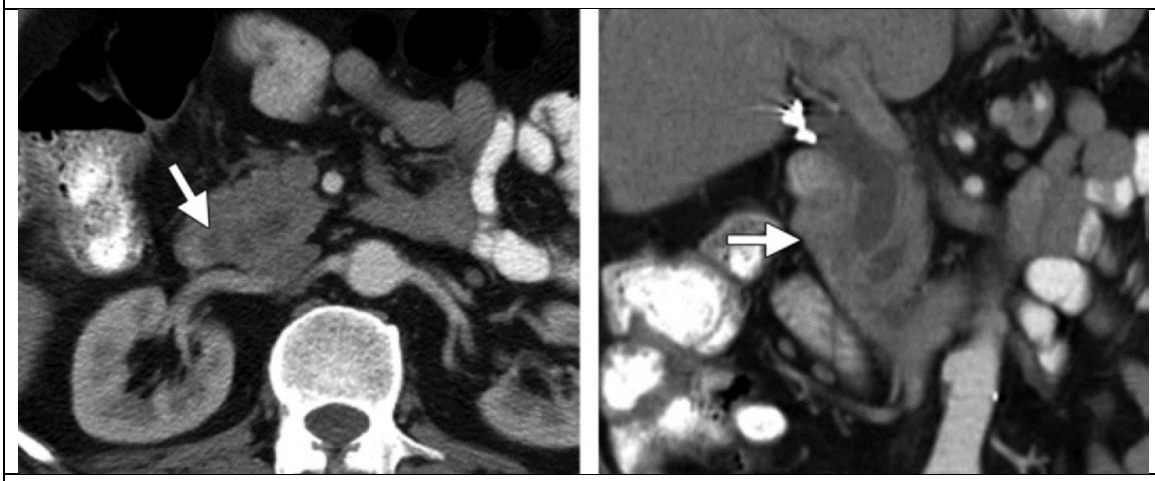
- Small nodular ampullary mass
- Duodenal papillary bulging
- Irregular asymmetric luminal narrowing of the distal CBD
- Diffuse upstream intra- and extrahepatic biliary dilatation and pancreatic duct



B-Distal CBD Cholangiocarcinoma



C-Duodenal Adenocarcinoma



D-Pancreatic head adenocarcinoma involving ampullary region

Pancreatic Trauma

- It is relatively uncommon
- 5% of abdominal trauma
- Usually caused by blunt trauma.
- FAST has a limit role in acute pancreatic injury.
- CT is the most reliable imaging modality.
- MRCP is useful in evaluating pancreatic duct disruption.

❖Type of Injury:

- In adults: Steering wheel impact in motor vehicle collision
- In children: bicycle handlebar impact is a common cause.
- In infants: Nonaccidental trauma is the major consideration

❖Site of Injury:

- Pancreatic injury typically results from severe anterior-to-posterior force vectors compressing the pancreas against the spine, with the injury commonly occurring just to the left of the mesenteric vessels.
    - **2/3 in the pancreatic body**
    - **1/3 in the head, neck, and tail.**

❖Scoring Pancreatic Injury

Grade	Description
I	Minor contusion or laceration without ductal injury
II	Major contusion or laceration without ductal injury or tissue loss
III	Distal transection or pancreatic parenchymal injury with ductal injury
IV	Proximal transection or pancreatic parenchymal injury involving the ampulla
V	Massive disruption of the pancreatic head

❖Imaging findings:

Pancreatic contusions:

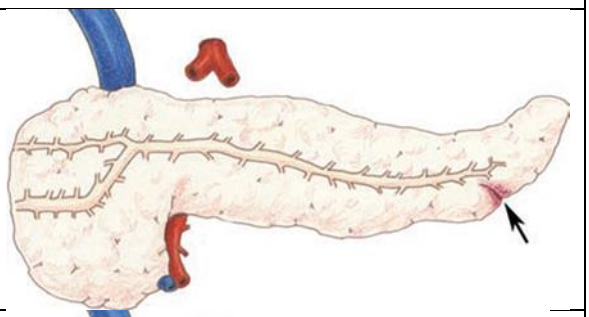
- Focal or diffuse areas of hypoattenuation surrounded by normally enhancing pancreatic tissue.

Pancreatic Lacerations:

- Areas of linear hypoattenuation perpendicular to the long axis of the pancreas.

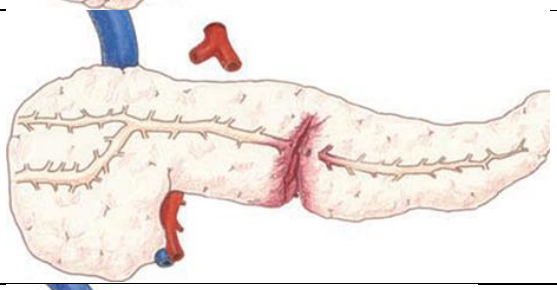
Superficial pancreatic laceration

- Involves <50% of the thickness of the pancreatic parenchyma
- does not involve the pancreatic duct.



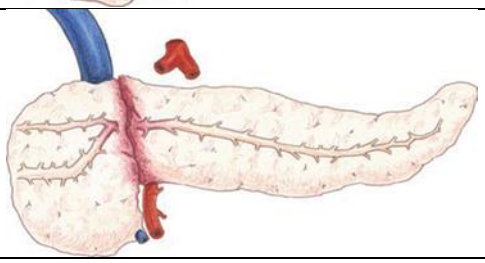
Deep pancreatic Laceration

- involves >50% of the thickness of the pancreatic parenchyma
- injury to the pancreatic duct.



Pancreatic Transection

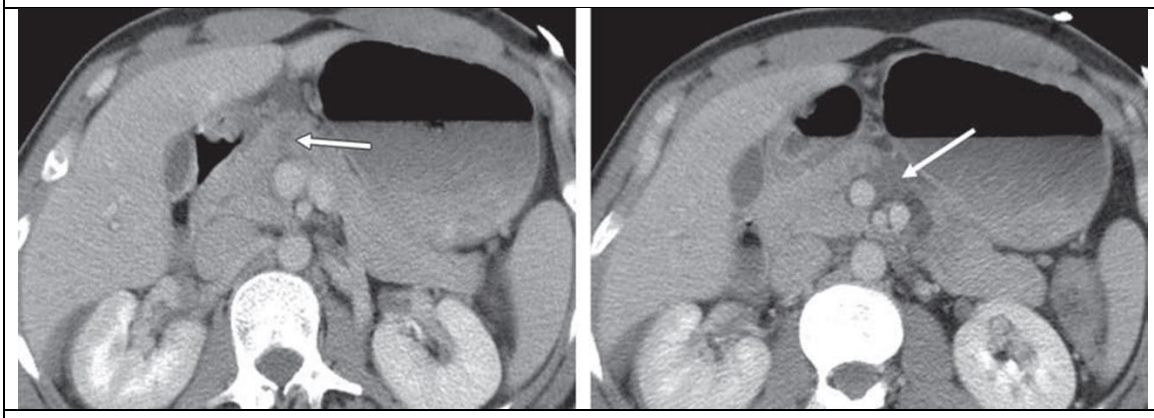
- involves the full thickness of the pancreatic parenchyma
- transection of the pancreatic duct.



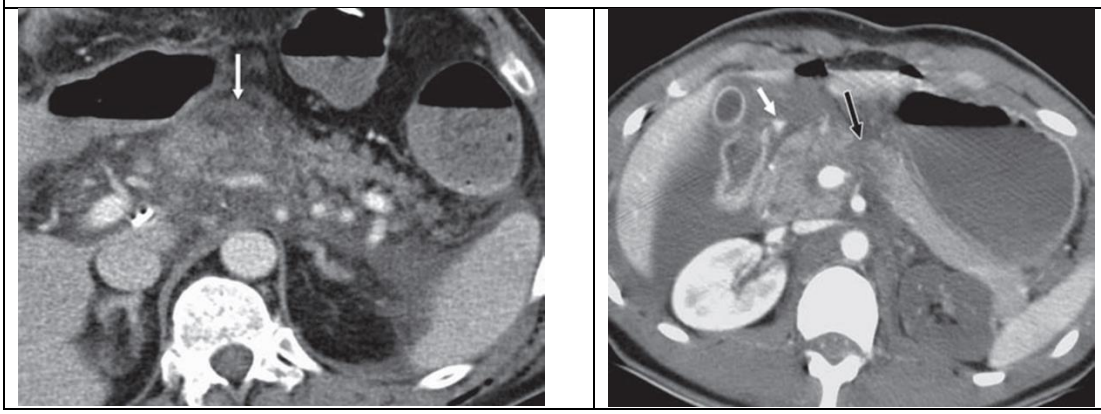
other imaging findings:

- Focal or diffuse pancreatic enlargement or edema.
- Thickened anterior pancreatic fascia and fluid in the lesser sac.
- Fluid rim between pancreas and splenic vein.
- Active bleeding or extravasation of intravenous contrast material.

Pancreatic Laceration



Pancreatic Transection



Spleen

❖ List of Contents:

- **Splenic Anatomy**
- **Imaging Techniques**
- **Congenital Anomalies**
- **Splenomegaly**
- **Focal Solid Lesions**
- **Focal Cystic Lesions**
- **Vascular Lesions**
- **Trauma**

Splenic Anatomy

- The spleen is the largest ductless gland
- The spleen is the largest single lymphatic organ in the body.

Site: Left Hypochondrium

Size:

- Thick = 2.5 cm
- Board = 7.5 cm
- Length = 12.5 cm

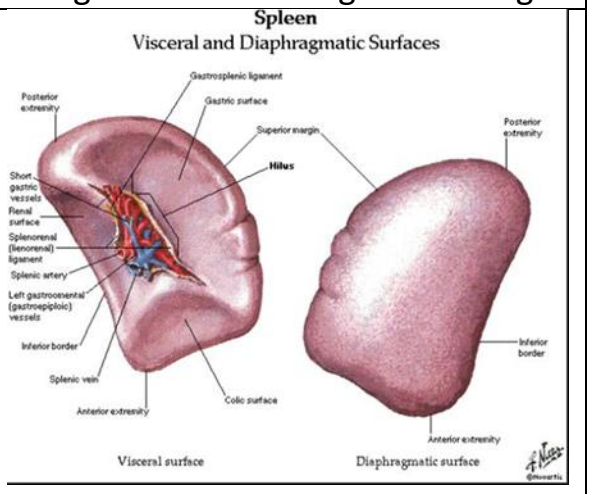
Shape: Wedge-shaped organ

Splenic functions:

- Immunologic surveillance
- Red blood cell breakdown.
- Splenic contraction for blood volume augmentation during hemorrhage

2 splenic surfaces:

- **Diaphragmatic surface**
- **Visceral surface:**
  - Anterior = Gastric ridge
  - Posterior = Renal portion
  - Pancreatic tail



2 histological compartments:

- The white pulp is made up of T and B lymphocytes
  - The red pulp is composed of rich plexuses of tortuous venous sinuses.

Imaging Techniques

❖ US

- Patient position: Supine, left lateral position
- visualized best obliquely in the 9th or 10th intercostal spaces
- Splenic echogenicity > liver echogenicity

❖ CT protocol

Standard CT:

- NCCT
- Portal venous phase (60-80sec)

Triphasic CT:

- NCCT
- Late arterial phase: 35 sec post injection of contrast
- Portal venous phase: 60-80 sec post injection of contrast
- Delayed phase: 3-5 min post injection of contrast

Normal CT features of spleen:

- CT attenuation of splenic tissue is homogeneous.
- NCCT: Splenic attenuation < liver attenuation by 5-10 HU
- Arterial phase: Striking wave-like, somewhat psychedelic, pattern of enhancement is a product of the unique splenic vascular anatomy
- Splenic parenchyma should be assessed in portal venous phase

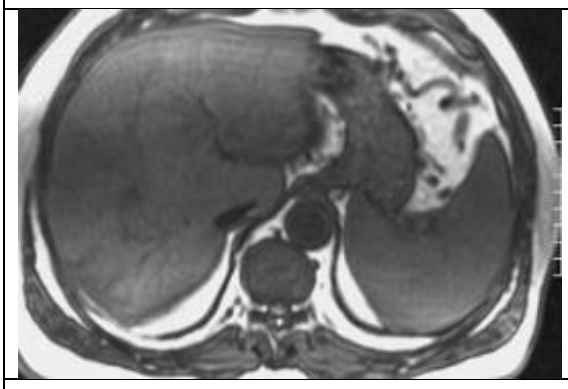
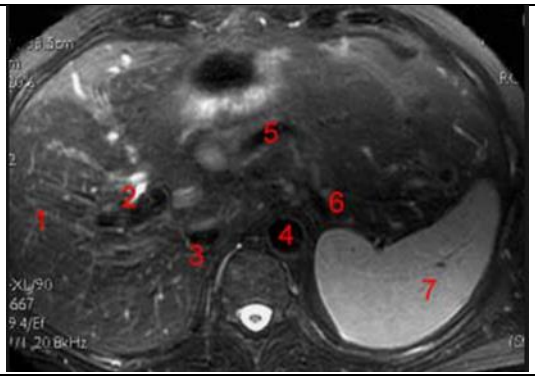
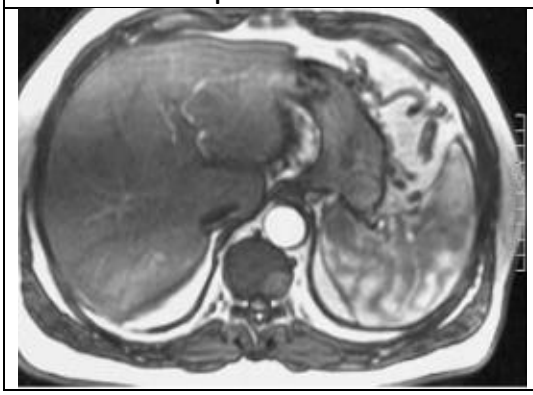
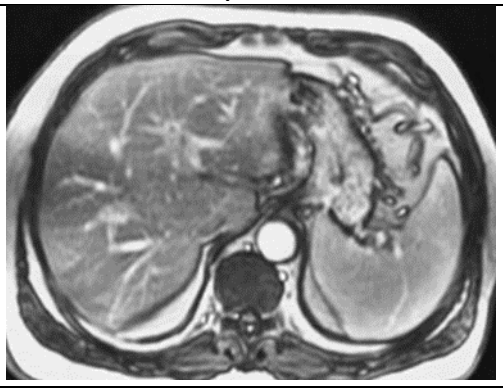
NCCT	Late arterial phase	Portal venous phase

❖ MRI protocol

- Axial T1 GRE in-phase
- Axial T1 GRE out of phase
- Axial T2 SSFSE
- Axial T2 FS
- Coronal T2
- Dynamic Axial T1 GRE

Normal MRI features:

- T1: Spleen is lower in signal than liver
- T2: Spleen is higher in signal than liver
- Arterial phase: alternating high and low signal intensity, resulting in a serpentine or arciform pattern
- Parenchymatous phase: Homogenous enhancement

Axial T1	Axial T2 FS
	
Late Arterial phase	Portal venous phase
	

Normal Variants and Congenital Diseases

1) Accessory Spleen = Splenuncului = Splenules

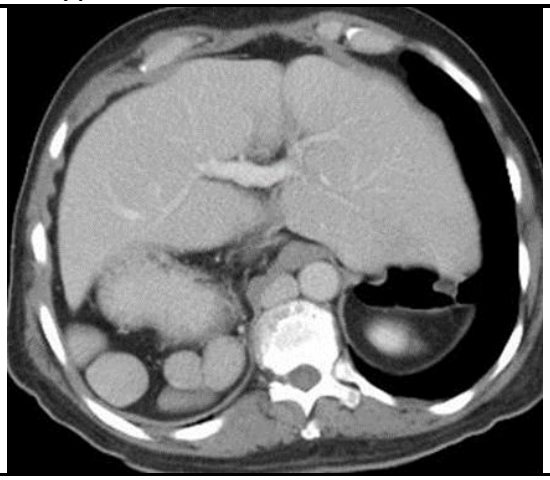
- 10% of individuals
- Solitary or multiple
- usually measure < 4 cm in diameter.
- Follows the CT attenuation, MR signal intensity and enhancement of the spleen on images obtained with various phases and pulse sequences

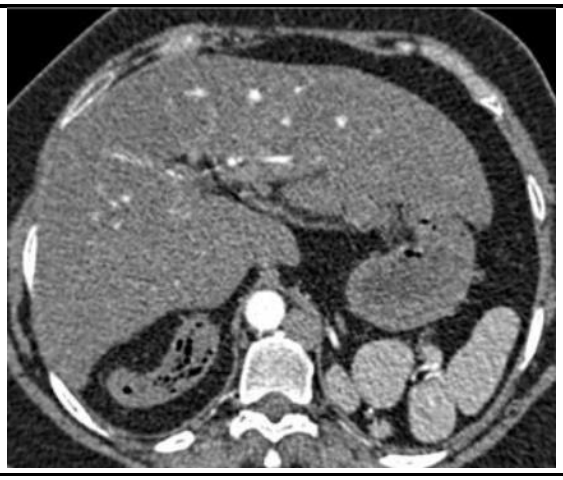
Location:

- Splenic hilum (most common)
- Gastrosplenic ligament
- Splenorenal ligament
- Pancreatic tail
- Greater omentum
- Small bowel mesentery
- Stomach or bowel wall
- Scrotum

2) Polysplenia

- Polysplenia is seen in association with abdominal situs and cardiovascular anomalies.
- This condition is more common in females.
- Classically, numerous small splenic masses are seen in the right or left hypochondrium





3) Wandering Spleen

- Wandering spleen is a rare condition in which the spleen migrates from its usual anatomical position, commonly to the lower abdomen or pelvis.

Splenomegaly

❖ Infection / Inflammatory Diseases

- **Viral**
  - EBV (infectious mononucleosis) - (common)
  - AIDS
  - CMV
  - Herpes simplex virus
  - Rubella
- **Bacterial**
  - Tuberculosis (miliary)
  - Subacute bacterial endocarditis
  - Typhoid fever
  - Brucellosis
  - Syphilis
- **Fungal**
  - Histoplasmosis (common)
  - Candidiasis
- **Parasitic disease**
  - Malaria (Common)
  - Schistosomiasis
  - Hydatid disease
  - Leishmaniasis kala-azar)
- Rickettsial
  - Typhus
- Sarcoidosis
- Amyloidosis

❖ Connective Tissue Disorders

- Rheumatoid arthritis (RA)
- Felty syndrome
- Juvenile rheumatoid arthritis (JRA)
- Still's disease
- Systemic lupus erythematosus (SLE)

❖ Portal Hypertension:

- **Pre-sinusoidal**
  - Portal vein thrombosis
  - Splenic vein thrombosis
  - Extrinsic compression of portal vein
  - Bilharziasis / Schistosomiasis
- **Sinusoidal**
  - Cirrhosis
- **Post-sinusoidal**
  - Budd-chiari syndrome
  - Hepatic veno-occlusive disease
  - Right heart failure or biventricular cardiac

❖ Metabolic Diseases:

- **Glycogen Storage Diseases**
- **Lipid Storage Diseases**
- **Mucopolysaccharidoses**

❖ Hematological Diseases

- **Anaemias**
  - Sickle cell disease
  - Thalassaemia
  - Hereditary spherocytosis
  - Pyruvate kinase deficiency
  - Thrombotic thrombocytopaenic purpura (TTP)
- **Neoplastic/proliferative/redistribution of haematopoesis**
  - Leukaemia (especially CML)
  - Polycythaemia rubra vera
  - Waldenström macroglobulinaemia
  - Myelofibrosis
  - Extramedullary haematopoiesis
  - Osteopetrosis
  - Idiopathic hypereosinophilic syndrome

❖ Neoplastic:

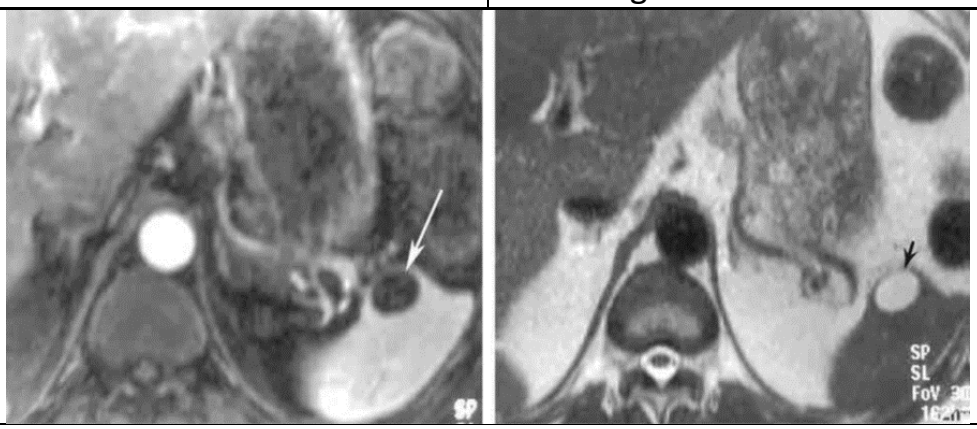
- **Lymphoma**
- **Leukemia**

Splenic Cystic Lesions

❖ **Developmental:** ○ **Splenic cyst** ○ **Lymphangioma**
❖ **Inflammatory:** ○ **Pyogenic abscess** ○ **Candidiasis abscess** ○ **Hydatid cyst**
❖ **Trauma: (see trauma)** ○ **Hematoma**
❖ **Neoplasms:** ○ **Cystic metastasis**

1) Splenic Cyst

- Well defined
- No enhancement
- Low T1
- High T2



2) Splenic Abscess

The main causes of splenic abscesses include

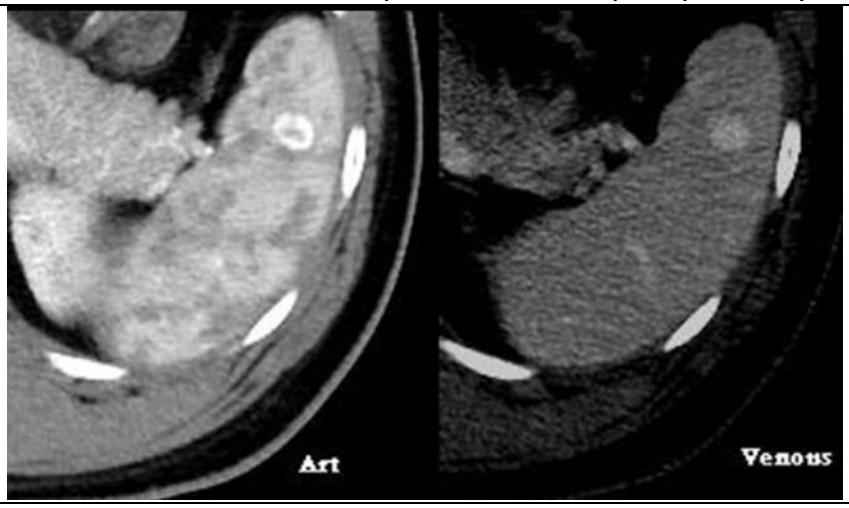
- Infection: anaerobes are thought to be most common infective agent
  - Metastatic infection
    - Blood bacterial dissemination, such as sepsis
    - Infective endocarditis
  - Continuous infection:
    - Perinephric abscess
    - Infected pancreatitis
- Splenic infarction and superimposed infection
- Trauma
- Immunodeficiency conditions (e.g. chemotherapy / transplant recipients, leukaemia and AIDS)

Splenic Solid Lesions

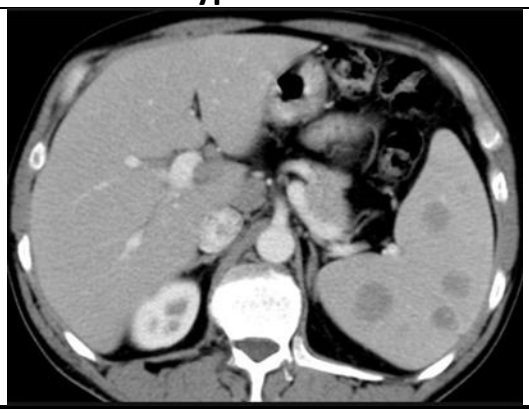
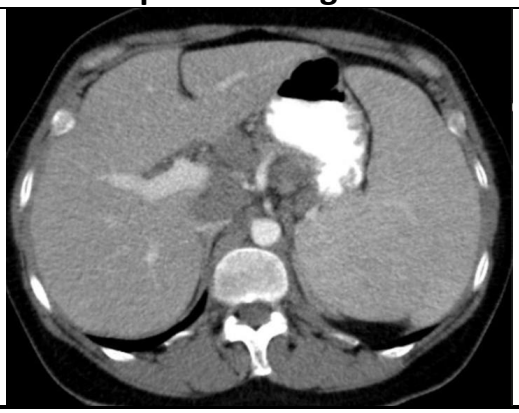
A- Benign: • **Hemangioma** • **Hamartoma**
B- Malignant: • **Lymphoma** • **Metastasis** • **Angiosarcoma**

1) Splenic Hemangioma:

- 2nd common focal lesion in the spleen after simple splenic cysts



2) Splenic Lymphoma:

Multifocal hypovascular lesions	Diffuse splenic enlargement
	

Splenic Vascular Disorders

- **Splenic Infarction**
- **Splenic Artery Aneurysm**
- **Splenic Vein Thrombosis**
- **Splenic Embolization**

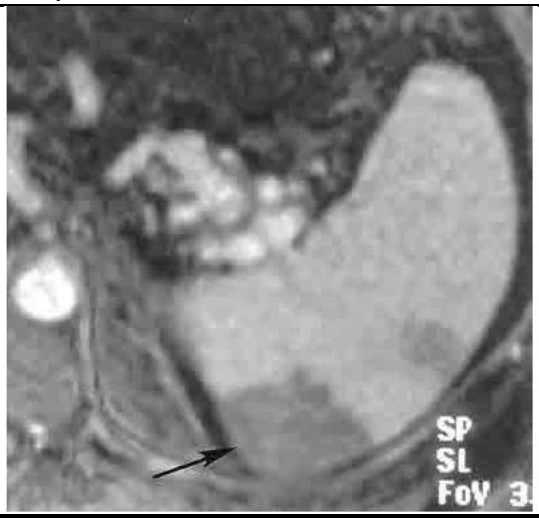
1) Splenic Infarction

Aetiology:

- Arterial emboli:
  - Sickle cell anemia, Gaucher disease,
  - Hematologic malignancies
  - Cardiac emboli
- Collagen vascular disease
- Portal hypertension.

Imaging patterns:

- Peripheral, wedge-shaped hypo-enhancing region: typical
- Multiple, heterogeneous areas of patchy enhancement
- Global splenic infarction, entire spleen is hypo-enhancing
- Splenunculus infarction



2) Splenic Artery Aneurysm & Pseudoaneurysm

- 1st common visceral arterial aneurysm
- 3rd common abdominal aneurysm (after the aorta and iliac vessels).

Aetiology:

- Atherosclerosis
- Congenital
- Mycotic
- Portal hypertension
- Fibromuscular dysplasia
- **Pseudoaneurysms from trauma and pancreatitis**

Imaging findings:

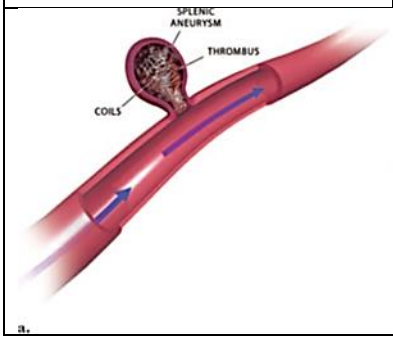
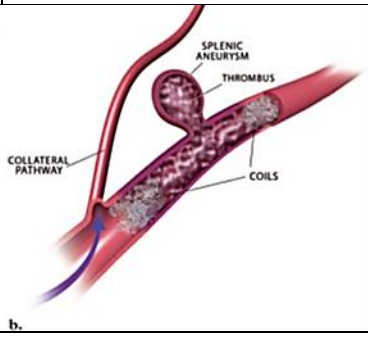
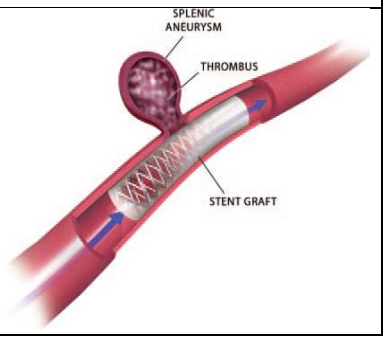
- Solitary 80%; Multiple in 20%
- Small (<2 cm in diameter)
- Saccular
- Most common location: at bifurcation of middle or distal segment of the splenic artery.

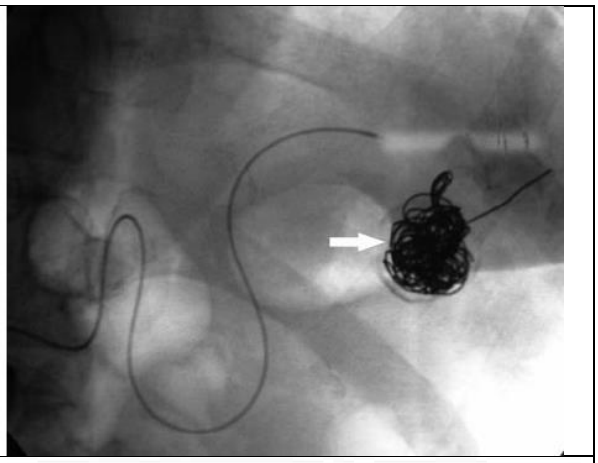
Indications for treatment

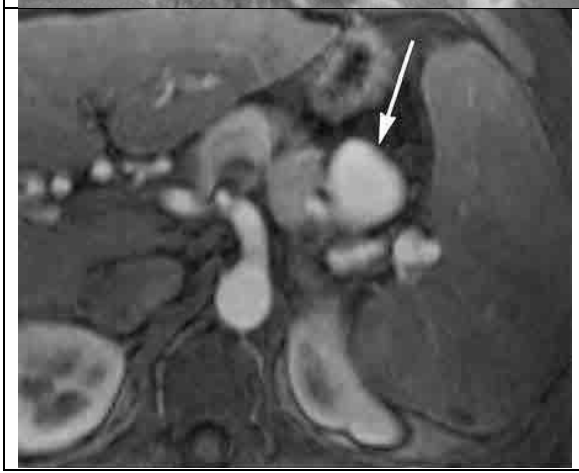
- Epigastric pain, left upper quadrant pain, back pain
- Female sex and childbearing age
- Presence of portal hypertension, planned liver transplantation
- Pseudoaneurysm of any size
- Aneurysm diameter > 2.5 cm.

Treatment options:

- **Surgical**
- **Transcatheter approach:**
  - Embolization of the aneurysm with multiple coils placed
  - Embolization of the splenic artery segment proximal and distal to the aneurysmal neck (the so-called sandwich method)
  - Complete exclusion of an aneurysmal sac with a stent-graft
- **Percutaneous needle approach:**
  - Administration of thrombin directly into the aneurysm

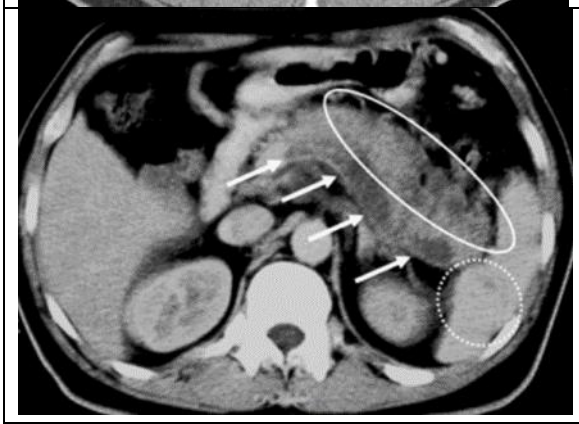
Embolization of the aneurysm with multiple coils placed	Embolization of the splenic artery segment proximal and distal to the aneurysmal neck = sandwich method	Complete exclusion of an aneurysmal sac with a stent-graft
		





3) Splenic Vein Thrombosis

- Splenic vein thrombosis has multiple causes
- Most commonly secondary to pancreatitis.

CT	Post contrast T1 GRE
	

4) Splenic Embolization

Indications of splenic Embolization:

1) Splenic trauma + hemodynamically stable
2) Hypersplenism (Splenomegaly + Thrombocytopenia + Leukopenia + Anemia)
3) Pseudoaneurysm: pancreatitis
4) Portal hypertension
5) Splenic steal phenomena

Complications:

- Splenic abscess
- Splenic rupture
- Septicemia
- Splenic vein thrombosis
- Unremitting bronchopneumonia

Embolic agents used:

- PVA
- Gelatin sponge
- Coils

2 approaches:

- Complete splenic arterial embolization
- Partial splenic arterial embolization
    - Selective: embolization of few distal branches to achieve complete stasis
    - Non-selective: embolization of main splenic artery to achieve reduced parenchymal blush.

Splenic Trauma

- The spleen is the most commonly ruptured intraabdominal organ.

Grade I:

- Subcapsular hematoma < 10% of surface area
- Capsular tear <1 cm in depth

Grade II

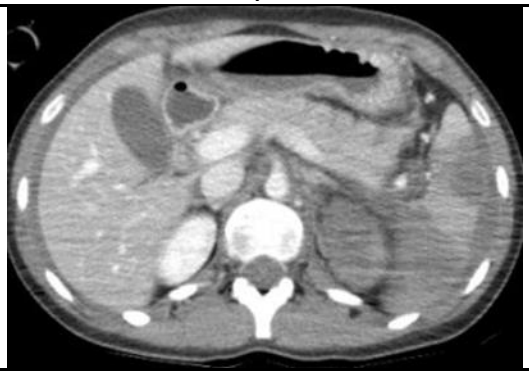
- Subcapsular hematoma of 10-50% of surface area
- Intraparenchymal hematoma < 5 cm in diameter
- Laceration of 1-3 cm in depth and not involving trabecular vessels

Grade III:

- Subcapsular hematoma > 50% of surface area or expanding and ruptured subcapsular or parenchymal hematoma
- Intraparenchymal hematoma > 5 cm or expanding
- Laceration >3 cm in depth or involving trabecular vessels

Grade IV

- Laceration involving segmental or hilar vessels with devascularization of > 25% of the spleen



Grade V

- Shattered spleen
- Hilar vascular injury with splenic devascularization.

Transplantation in Abdomen

I- Liver Transplantation:

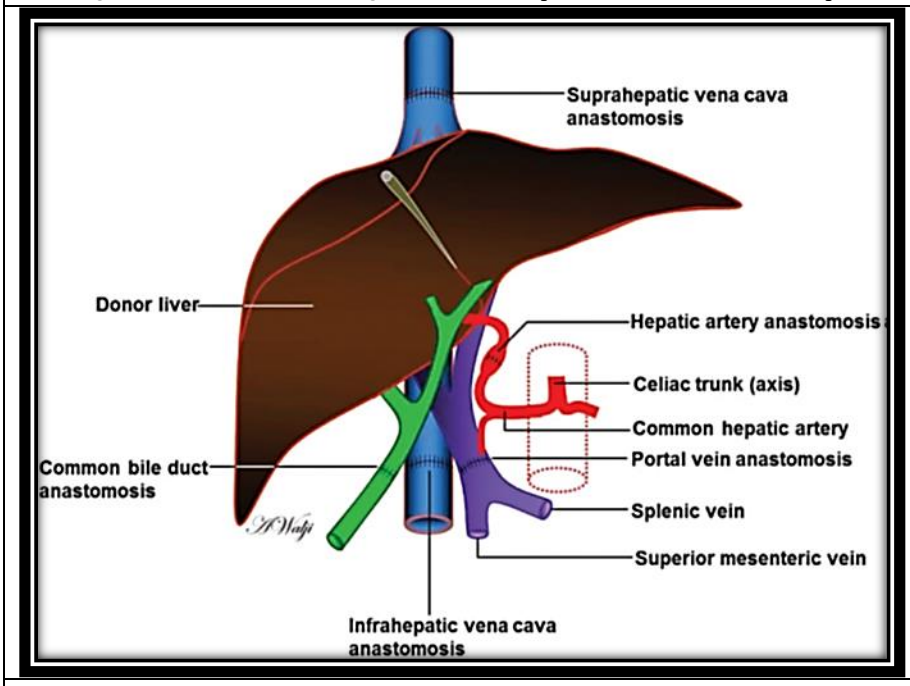
❖ Indications of Liver Transplantation

Condition	Indications
Alcoholic cirrhosis	Patients must abstain from alcohol for 6 months, undergo treatment for alcohol abuse, and have a strong psychosocial support system
Chronic hepatitis	Hepatitis B, C, and D infection; autoimmune hepatitis; chronic drug use; cryptogenic cirrhosis; chronic active hepatitis
Primary hepatic tumors	Hepatocellular carcinoma (grade 1 or 2, <5 cm, and no extrahepatic metastases or macrovascular invasion)
Cholestatic diseases (intrahepatic and obstructive)	Primary biliary cirrhosis, sclerosing cholangitis, cystic fibrosis, biliary atresia, Caroli disease, Alagille syndrome, Byler disease, familial cholestatic syndromes
Fulminant liver failure	Drug toxicity, toxins, viral hepatitis infection
Metabolic disease	α-1-antitrypsin deficiency, glycogen storage disease, Wilson disease, hemochromatosis*
Other conditions	Budd-Chiari syndrome, parenteral nutrition–induced cirrhosis, trauma

❖ Contraindications for Liver Transplantation

Absolute
Active uncontrolled infection or sepsis
Brain death
Active alcohol or substance use
AIDS
Extrahepatic malignancy
Severe cardiopulmonary disease
Inability to comply with a medical regimen
Lack of a psychosocial support system
Anatomic abnormalities that preclude liver transplantation
Compensated cirrhosis with no complications
>25% fatty infiltration
Relative
Human immunodeficiency virus infection
Cholangiocarcinoma*
Advanced age
Psychologic instability

A) Whole Liver (Orthotopic Liver Transplantation) (OLT)



Most common procedure in adults

1) Hepatic arterial anastomosis:
   - End-to-end anastomosis between either the donor celiac axis or the donor common hepatic artery–splenic artery branch point and the recipient proper hepatic artery (either the gastroduodenal artery bifurcation or the left-right hepatic artery bifurcation).
2) Portal vein anastomosis:
   - End-to-end anastomosis between donor and recipient portal veins
3) IVC anastomosis:
   - Infrahepatic end-to-end anastomosis
   - Suprahepatic end-to-end anastomosis
4) Biliary anastomsosi:
   - End-t-end anastomosis between donor and recipient's CHD

B) Split Liver Transplantation:

- Dividing a cadaveric liver:
  - Lateral segment of the left lobe may be transplanted into a pediatric patient
  - The remainder of the liver may be transplanted into an adult.

C) Living donor liver transplantation (LDLTs):

- Account for approximately 10% in adults.

II- Pancreatic Transplantation

❖ The American Diabetes Association 2006 Indications for pancreas transplant are:

- Hyperlabile diabetes defined by frequent acute severe metabolic complications (hypoglycemia, marked hyperglycemia, and ketoacidosis) requiring medical attention
- Clinical and emotional problems with insulin therapy that are incapacitating
- Consistent failure of insulin based management to prevent complications
- Presence of (two or more) diabetic complications that are progressive and unresponsive to intensive insulin therapy
- Early diabetic nephropathy
- Proliferative retinopathy
- Symptomatic peripheral or autonomic neuropathy
- Vasculopathy with accelerated atherosclerosis

The indications for a pancreas and kidney transplant are:

- CKD stages 4 or 5 (creatinine clearance <30 ml/min) with type 1 diabetes and with other diabetic complications
- Prior renal transplant which is failing in a type 1 diabetic

The most common types of pancreas transplants are:

- **SPK:** simultaneous pancreas and kidney transplant (~78% of cases)
- **PAK:** pancreas after kidney transplant (~16% of cases)
- **PTA:** pancreas transplant alone (~7% of cases)

❖ Surgical Technique & Anatomy:

Arterial Supply:

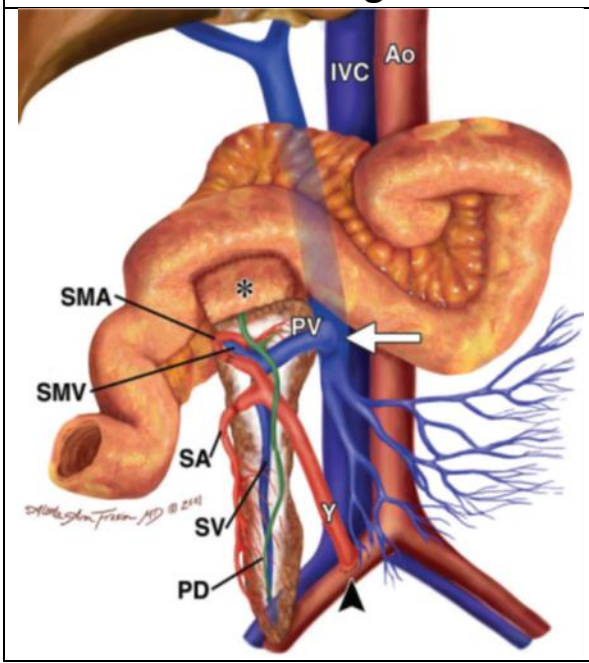
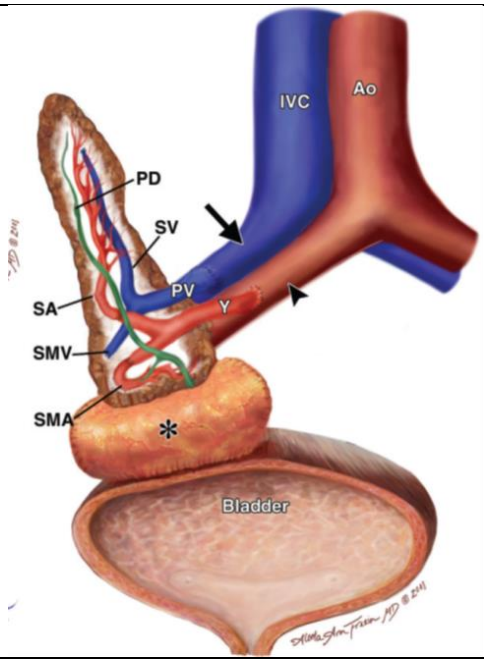
- The donor common, internal, and external iliac arteries are attached to the donor SMA (supplies head of pancreas) and splenic artery (supplies pancreatic body and tail), forming a Y graft. The common iliac artery portion is then anastomosed to the recipient common iliac artery or external iliac artery

Venous drainage:

- Anastomosis between the donor portal vein, which functions as the main graft vein, and the recipient SMV or iliac vein

Exocrine Secretions

- The donor duodenum containing the ampulla of Vater is harvested with the donor pancreas.
- Pancreatic exocrine secretions may be drained to the gastrointestinal tract (enteric drainage) or the bladder (bladder drainage) via the donor duodenum.

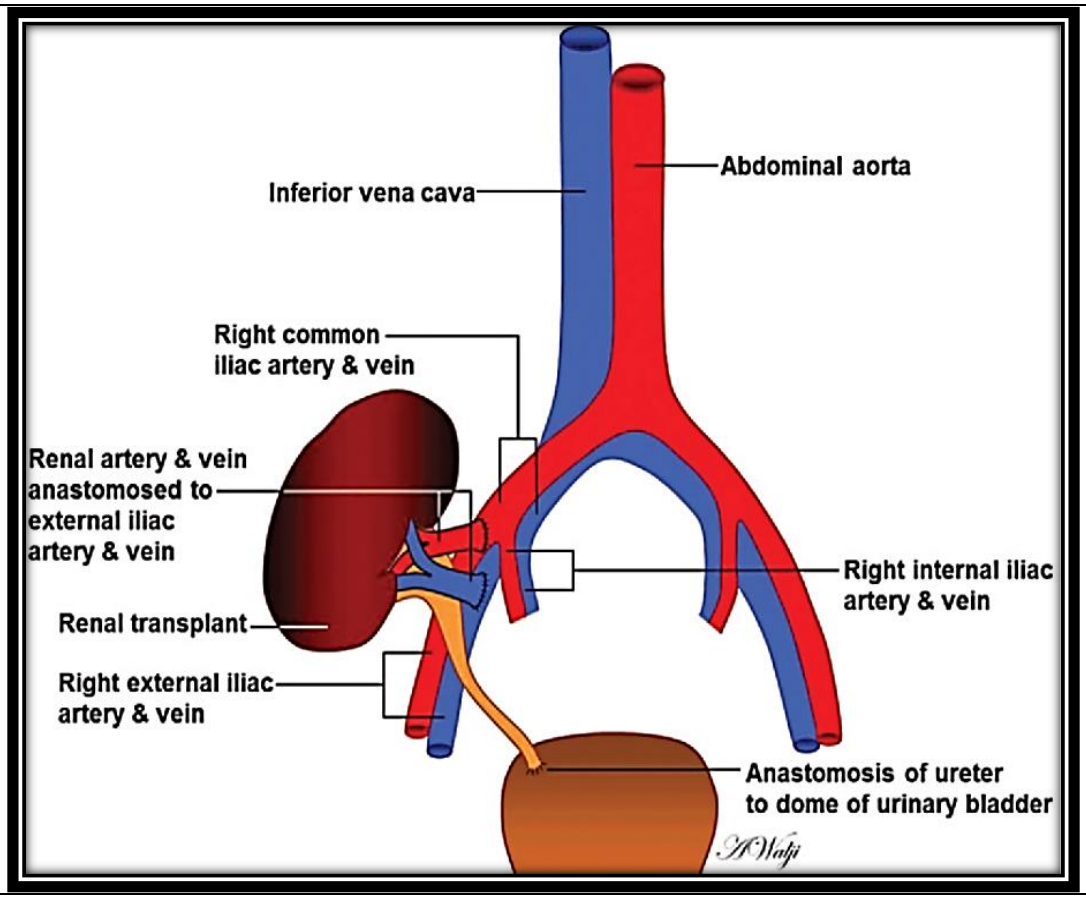
A- Enteric Drainage	B- Bladder Drainage
	

III- Renal Transplantation

❖ Indications:

- Any condition that leads to **end-stage renal disease**

❖ Surgical Technique & Anatomy:


Inferior vena cava
Abdominal aorta
Right common iliac artery & vein
Renal artery & vein anastomosed to external iliac artery & vein
Right internal iliac artery & vein
Renal transplant
Right external iliac artery & vein
Anastomosis of ureter to dome of urinary bladder
A Walji

Renal transplant:

- is placed extraperitoneally within the iliac fossa RT > LT

Arterial anastomosis

- End-to-side anastomosis between the donor renal artery and the recipient external iliac artery.

Venous anastomosis:

- End-to-side anastomosis between the donor renal vein and the recipient external iliac vein.

Ureteroneocystostomy

- Implantation of the donor ureter into the bladder dome.

❖ Pre-operative Evaluation of Living Donor

- Evaluation of parenchyma
- Volumetric study
- A= Arterial anatomy and variants
- V= Venous anatomy and variants
- P= Portal anatomy and variants
- B= Biliary Tree anatomy and variants

❖Post-Transplantation Complications

- Fluid Collections:
  - Seroma, hematoma, lymphocele, urinoma, or pseudocyst
  - Abscess
  - Pleural effusion
  - Minimal ascites
- Immunological complications:
  - Graft Rejection (1st most common cause of graft failure)
- Vascular complications:
  - Venous thrombosis (2nd most common cause of graft failure).
  - Arterial thrombosis
  - Arterial stenosis
  - Pseudoaneurysm
  - Arteriovenous Fistula
- Biliary Complications (in Liver Transplantation):
  - Biliary leak: Biloma – Biliary Peritonitis
  - Biliary stricture / obstruction
- Bowel Complications (in Pancreatic Transplantation):
  - Bowel obstruction
  - Anastomotic leak
  - Bowel infection: pseudomembranous or cytomegalovirus colitis
- Urinary Complications (in Renal Transplantation):
  - Urinary leak
  - Urinary obstruction / ureteric stricture: Hydronepohrosis
  - Vesico-ureteric reflux
- Infection: Pancreatitis – Pyelonephritis – Sepsis
- Post-Transplantation Lymphoproliferative Disease (PTLD)

I- Graft Rejection

- Graft rejection continues to be the primary cause of graft loss
- 5% - 25%, depending on the immunosuppressive regimen

a) Hyperacute rejection:

- Develops immediately after transplantation
- Response to the presence of preformed circulating cytotoxic antibodies in the recipient's blood.
- It causes thrombosis and immediate graft loss.

b) Acute rejection:

- Develops 1 week to 3 months after transplantation
- Autoimmune vasculitis, which results in small-vessel occlusion and decreased perfusion, leading to infarction if severe and untreated.

c) Chronic rejection

- remains the major long-term cause of graft failure
- develops after the first 6 months
- Manifests as an insidious loss of exocrine and then endocrine function secondary to small-vessel endarteritis, acinar atrophy, and interstitial fibrosis
- Fibrosis and atrophy of the gland, resulting in insidious, progressive loss of graft function

Percutaneous biopsy is the standard for diagnosis and grading of rejection.

US (Nonspecific):

- Graft enlargement
- Increased heterogeneity of the parenchymal echotexture

Color Doppler US:

- Major vessel patency, it will help exclude graft thrombosis as a cause of poor function but cannot help differentiate pancreatitis from rejection

MRI:

- T2: edema and increased signal intensity (Non-specific)

II- Vascular Complications

1) Arterial Thrombosis:

- 3m after transplantation
- Most severe vascular complication => Graft dysfunction and necrosis
- Duplex => Absent signal in the graft artery
- CTA/MRA => Intraluminal filling defect in the graft artery
- Scintigraphy => Absent tracer uptake

A- Hepatic Artery Thrombosis:

10%adults - 20%Pediatric - 60% of vascular complication
=> Biliary ischemia:
* Biliary stricture (anastomotic and non-anastomotic) => US/MRCP: Biliary dilatation
* Biliary leak => Biloma / Biliary Peritonitis => HIDA scan: Extrabiliary tracer polling
* Biliary abscess => 2ry infection of biloma
* Biliary Cast syndrome => Intaluminal cast like material => Obstruction

B- Renal Artery Thrombosis:

1% of renal transplants
DTPA => Absent tracer uptake by whole or part of the transplanted kidney.
=> Ureteric Ischemia:
* Ureteric stricture (Vesicu-uretric junction) => Proximal hydrouretro-nephrosis
* Urinary leak => Urinoma => CTU/MRU: Contrast extravasation outside urinary system

C- Splenic a. / SMA Thrombosis:

10% of pancreatic transplantation
* Graft dysfunction & necrosis
* Pancreatitis
* Sepsis

D- Intestinal Transplant:

10% of intestinal transplantation
* Bowel infarction => No wall enhancement + Intestinal wall gas + Mesenteric & Portal vein gas
* Bowel stricture
* Anastomotic dehiscence
* Intestinal dysmotility and infections.

2) Arterial Stenosis Duplex:

Normal:

In first 72 hrs => Reperfusion edema = RI>0.8

then return to normal RI = 0.5 - 0.8

At stenosis:

* PSV >200 cm/sec
* RI > 0.8
* Absence of forward diastolic flow
* Post stenotic turblent flow

Intraparenchymal:

* Low RI <0.5
* Long systolic acceleration time (SAT >0.08 sec)
* Tardus-parvus waveform distal to the stenosis

3) Venous Thrombosis

Duplex US

- complete thrombosis: Absence of signal in the presence of.
- Partial thrombosis: nonocclusive filling defect.

4) Arteriovenous Fistula (AVF)

- Etiology: Biopsy or percutaneous interventional technique performed on the transplant allograft.
- Duplex:
    - Focal area of aliasing
    - High-velocity, low-resistance feeding artery
    - Pulsatile “arterialized” draining vein
- Large AVF may cause graft ischemia and dysfunction due to arterial “steal” phenomenon

5) Pseudoaneurysm

- Aetiology: caused by infection, surgery, percutaneous interventional procedures, or biopsy-related trauma.

Duplex US:

- Focal saccular arterial outpouching with swirling flow within the pseudoaneurysm sac and bidirectional flow in the neck

III- Post-Transplantation Lymphoproliferative Disorder (PTLD)

- Uncommon complications of transplantation.
- The majority of PTLDs arise within 1 year of transplantation.
- The majority of PTLDs are B-lymphocyte proliferations

a) Abdominal Manifestations of PTLD:

Liver:

- Multiple discrete nodular low attenuation lesions
- Infiltrative pattern, consisting of a geographic or an ill defined region of low attenuation with or without hepatomegaly
- Periportal mass that extends into the biliary tree with associated periportal lymphadenopathy.

Spleen:

- Splenomegaly
- Multiple focal low-attenuation lesions.

Renal:

- A heterogeneous mass surrounding hilar vessels
- Multifocal parenchymal renal masses

Adrenal:

- Diffuse infiltration with glandular enlargement
- A homogeneous low-attenuation mass

Hollow organ viscera:

- Localized circumferential wall thickening + Dilatation of involved loops
- Luminal excavation or ulceration
- Eccentric polypoid masses
- Extramural extension
- Intussusception.

Lymph node:

- Abdominal LNs
- Retroperitoneal LNs

Pancreatic:

- Diffuse glandular enlargement
- Focal pancreatic mass

b) Thoracic Manifestation of PTLD:

- A pulmonary mass and nodules, either solitary or multiple, are the most common thoracic manifestation of PTLD.
- Lymph node enlargement
- Airspace consolidation
- Pleural effusion
- Pericardial effusion
- Thymic enlargement
- Chest wall soft-tissue involvement

c) CNS Manifestations of PTLD:

- Periventricular lesion + hemorrhage/necrosis + Peripheral enhancement.

d) H&N Manifestations of PTLD:

- Waldeyer ring (85% of cases)
- Cervical lymphadenopathy (40%)
- Less common manifestations include orbital and sinonasal disease

e) Musculoskeletal Manifestations of PTLD:

- Very rare

❖ Role of Intervention Radiology:

A- Catheter Drainage:

- Diagnostic and therapeutic purposes in fluid collections such as abscesses, hematoma, biloma

B- Vascular Interventional:

- Angioplasty for hepatic artery stenosis
- Coil embolization of or stent placement for pseudoaneurysms,
- Angioplasty or stent placement for all venous stenoses.

C- Biliary Intervention:

- Angioplasty (with or without stent placement) across strictures
- Percutaneous transhepatic biliary drain (PTD) across strictures
- Stent placement across bile leakage sites
- Catheter drainage of bilomas
- Removal of occluded biliary stents.